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TRANSACTIONS

OF THE

THIRTY-SECOND ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

HELD AT

DETROIT, MICH.,

SEPTEMBER 12-17, 1927

Academy
gift
1-9-1928

The Thirty-third Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at St. Louis, Mo., October 15-19, 1928. The attention of the members is called to the ruling of the Council, that when a paper published in the Transactions is illustrated, the author must pay the total cost of all colored plates, and one-half the cost of all other cuts. The Editor will furnish an estimate of the cost of such cuts and plates. (*All papers, with illustrations, must be handed to the Secretary immediately after they are read.*)

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TRANSACTIONS

OF THE

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JOINT SESSION

PRESIDENT'S ADDRESS.

A SERMON.

ROSS HALL SKILLERN, M.D.

PHILADELPHIA, PENNSYLVANIA.

"Age cannot wither nor custom stale their infinite variety."

This discourse will refer to no Shakespearean heroine of the Nile, but rather to certain operations as now practiced upon one of the accessory sinuses of the nose, and incidently to certain human emotions and their phases which directly concern the doctor of laryngology, particularly in his relations to himself, his colleagues and incidently to the laity at large.

Speaking of operations, did it ever occur to you that there is no standard or set procedure for certain definite pathologic entities, so to speak, that may occur in these cavities? It is true, that in the event of an ethmoidal suppuration, we have a number of methods immediately at hand, such as the Hajek, Mosher, Sluder, Ballenger, etc., etc., but which one is indicated for that particular case? Again, a frontal sinus may present itself requiring some form of an external operation. What method is applied? If the patient happened to be in Germany, a Killian would unhesitatingly be performed; if in France, some modification of the Luc; if in England, one of the methods advocated by Tilley or Watson Williams; while here, one of these foreign procedures as modified by that particular operator. The only purely original American method that we have, appears to be one devised by a general surgeon, Lothrop, and so far as I know is only used in a modified form by the American sinuologist. Now how did all this come about? Why do we not have a systematized procedure in the application of surgical technic in the sinuses as well as, we will say, in the upper abdomen? I think that this can be answered by contemplating the lack of uniform teaching in the postgraduate schools and courses in the United States. Each individual teacher here has usually received his training from one or more European clinics, and is naturally prone to

follow the teachings of a certain particular outstanding authority, who had always been a hero in his eyes. It follows that his students in turn will be schooled in the belief of that particular Hofrat or professor, and therefore will have a decided English, German or French leaning, as the case may be. Now, in passing, this brings the thought of our relations to foreign countries, notably the German speaking ones, so far as our specialty is concerned. Gentlemen, the time has passed when we were subservient to the teachings and methods which had there emination in these sources. A pilgrimage to these shrines lasting but a few weeks, compared to the available well rounded postgraduate courses of some of our own Universities, is like sending a boy to a man's work: he can attempt it, but what he accomplishes is only a smattering and usually badly done at that. Reverting again to the lack of uniformity in sinus technic, other influences besides teaching have crept in, such as custom, precedence, ability, ignorance and even intolerance. Regarding custom, precedence has been all too slavishly followed. Suppose we continued the teachings of our predecessors in their originality, how absurd certain things would seem. For instance, Charles II had a law enacted which prohibited coaches from the streets of London on account of the destruction caused by their wheels to the paving stones. So do the trucks nowadays, but instead of abolishing the trucks, we concrete the roads. Again, coming down to our own time, who does not remember when everybody drank from the same cup at the public fountain, when we rode in street cars pulled by horses, and nobody took a bath until Saturday night. But you say all this has changed in the scientific light of a new era. Very well, let us see! Since the origin of the species, the prehistoric man, meeting with another and not wishing to fight, dropped his weapon and extended his right hand as a symbol of friendliness. This original purpose has long since passed into forgetfulness, but it is still the custom to shake hands on meeting a friend or acquaintance. Another instance; in the dark ages medicine and magic were almost synonomous. When a patient consulted the doctor-magician, it was a custom for the medical man to place his lips on the part that was ailing and suck out the evil. We all know how to this day a bruised finger is instantly placed in the mouth and sucked, and the old nursery remedy of kissing it and making it well no doubt

descended from this same origin. Scratch the epiderm of the civilized man and the barbarian is found in the derm.

Now let me cite another example in close relation to medicine, erysipelas, which still sometimes occurs as a complication of purulent sinusitis, but less and less in evidence owing to better asepsis in our methods of treatment. Formerly it was considered not only a complication but an etiologic factor of no little importance, and even Hajek, in a recent edition of his well known monograph, mentions it with more than passing interest. Here is another illustration of the changing order of things, for as late as the seventeenth century, St. Anthony's fire took its toll of victims were they prince or pauper. It is curious to recall that this disease received its name of St. Anthony's fire because sufferers from it were apt to be cured after being treated by the wise monks at the Abbey of St. Antoine at Vienna. St. Anthony of Padua had nothing to do with it, as is so commonly believed.

Again, common table salt would seem to be the last substance in the world around which one would associate romance or superstition, and yet this substance has been held in the highest esteem since the prehistoric era. The Israelites at the time of Moses did not believe proper digestion could occur unless salt were taken with their meals. At one time during the Graeco-Roman period, officials and employees of the State received a portion of their pay in salt, which was termed salarium, from which sprang our word salary. In medicine, the application of salt to a wound or sore is still in vogue, and even its accidental spilling has long since been accounted an omen of ill luck, originating perhaps in the tradition, that at the Last Supper, Judas, while in the act of presenting salt to the Master, spilled some upon the table. An old saying has it "When a man once comes to believe a thing, he is loath to discredit it", and habits are even more enduring that beliefs.

Let us now consider our discourse from the standpoint of personal ability. Some of us are blessed with a superabundance of natural ability, but most of us have to acquire it thru continued practice, observation and personal contact with men noted for their skill along given lines. I of course refer to operations and operative technic. The first requisite for a given operation is proper and suitable instruments. I recall the first course I attended in operative work on the

sinuses of a cadaver in Vienna, where the only instruments available were those ordinarly seen in the dead house. Naturally, the operations, like Gay-Lussac's law governing gases, were directly as to the conditions of the instruments. One cannot make pancakes without batter, neither can one perform an operation properly without suitable tools. For this very reason it is proper that in the beginning we select certain instruments that appeal to us, and practice with them (preferably on the cadaver) until their feel becomes almost of a second nature. How many of us, visiting clinics in other cities and noting with what ease a certain man performed a difficult operation, have bought his instruments and at the first opportunity, after arriving home, tried them out with results that were anything but pleasing. Now this miscarriage of technic did not result because operator A was necessarily more skillful than operator B, but because B did not have the same mastery of the procedure as A, having never before applied it. The chances are that A in the beginning had quite as much trouble as B, and only by constant study and application was he able to master it to such a degree as to make a seemingly difficult method look comparatively easy. So while there is a certain beneficient aspect toward the unusual and spectacular, nevertheless it is after all the end results that count, so that if operator B by his method procures the same result as operator A by his novel, but more or less involved procedure, there exists no reason (other things being equal) for one to claim superiority over the other, both in the final analysis having been to a like degree successful. And yet we cannot assume this attitude to such a degree that we become intolerant of another's views or methods. Intolerance has played a continuous role since the early dawn of history. Moses was denounced and threatened with violence when he first tried to free his people from Egyptian slavery. Socrates for his philosophic truths was given the cup of lethal hemlock, and you all know what happened to the Man of Galilee when he preached peace and good will to all men; He was taken out and nailed to a cross. Intolerance has no place in Medicine and never should have had, altho in the middle ages our profession subsisted upon it, traces of which remain to the present day. When new theories are advanced or new procedures recommended, many of us are prone to give heed with half an ear, so that when asked an opinion regarding

them, are apt to reply that we do not think much of them or that we do not do this form of operation. The world little cares what we don't think or what we can't do, but the world, particularly the medical profession at large, is vitally interested in what we do think and what we can do. Respecting this, how much better it is to be a positive and constructive man, everyone knowing where he stands, than a negative or indifferent one, whose beliefs and tenets are unconvincing and whose actions leave much to be desired.

Collective certitudes are infinitely superior to individual platitudes. This applies to operations as well as observations: an operative procedure that has received the stamp of approval collectively, so to speak, is worth a hundred methods that have only the support of single individuals, mostly their originators, to recommend them.

Now a word regarding the intranasal operations under local anesthesia, particularly those relating to the ethmoid. I have always been a rabid advocate of spending all the time necessary in procuring a thoro anesthesia of the parts before making the first incision. From my observations in many clinics in perhaps as many different lands, I have long since been convinced of the great wisdom of this principle. In the first place, while you have assured the patient that there will be no pain, nevertheless if he is human he has not been entirely convinced, and is apt to be a bit "jumpy" until he finds, that after your thoro anesthetization, the attacks upon his bulla or middle turbinate, as the case may be, are really painless. He will then be possessed of an assurance, and the operation will usually be concluded in peace and quiet. If, however, the cocainization is insufficient or but partially covers the operative area, and we have an apprehensive, neurotic individual to deal with, at the first stroke of the knife there will be much noise, some confusion and nothing accomplished. The more thoro the preparation of the parts, the more quiet the patient and the more successful the operation. Show me a case in which the anesthesia has been imperfect, and I will show you an imperfect operation. This adage may not hold good in every case, but if you will take the trouble to make a thoro examination where it has occurred, even if it is one of your own cases, I think you will bear me out in this assertion.

Now coming to the operation itself: If, when contemplating the advisability of an operation in a certain obscure case, the thought comes to us "That which the fountain sends forth returns again to the fountain", it may very materially change our viewpoint in that particular case. To deliberately rip out an ethmoid capsule for the purpose of removing the source of an unlocated suppuration in the nose, is like putting one's hand into a basket of snakes to find an eel. You may be successful, but the chances are small; furthermore, you may get your hand badly bitten into the bargain. Even tho we find to our own satisfaction that pus is continually coming from a certain portion of the ethmoid, constantly appearing in the same place after wiping away, we must not think of it as an abscess, but rather as a product and not the actual cause of the disorder, so that the mere evacuation will have little influence upon the trend of the infection unless adequate aeration and drainage is at the same time effected. Regarding the operation itself, I am inclined more and more toward conservatism, especially in those cases where little surgery has as yet been attempted. The removal of the middle turbinate with daily applications of a strong colloidal silver solution on tampons placed up and against the ethmoid capsule, which are permitted to remain for several hours, will ofttimes bring order out of chaos and obviate the necessity of further surgical procedures. If, instead, we should meet with one of those cases which has undergone repeated operations, and the entire ethmoid capsule is one disorganized suppurating mass, of course only a complete exenteration beginning with the uncinate process will suffice. I am inclined, however, to feel toward the socalled complete exenteration of the ethmoid as Voltaire said regarding the Holy Roman Empire, namely that it was neither Holy nor Roman nor was it yet an Empire, so the complete intranasal exenteration, while indeed internal, is neither complete nor is it an exenteration.

And now, in conclusion, a word to our younger brethren, altho to some of us older ones it might also not come amiss, and that is sticking to our ideals. Every man that reads medicine must sooner or later come to the conclusion that he is about to embark on a career that is a profession and not a business, a career in which the ideals are diametrically opposite to the accepted tenets of modern business. In the profession, the end results concern themselves primarily with

the final condition of the patient and secondarily with the remuneration received, while in business, the final analysis rests entirely upon the credit side of the ledger, regardless of the fate of the house with which the trading has been done.

The younger man starting out with ideals along these lines will soon find himself sorely beset, not only from those who are openly hostile, but from sources which he feels have only his best interests at heart. I recall only too well, some twenty odd years ago, a young man, having spent nearly two years abroad, returning from Vienna full of enthusiasm and with what he thought a message. This message was the recognition and treatment of accessory sinus disease. After much preparation and forethought, the evening came and the message was delivered. Instead of a lively discussion following, it was received in cold silence; and after the meeting had adjourned, one of the older men approached the essayist, and putting a warning hand on his shoulder said, "My boy, if you don't stop sticking those needles into people's antrums you are going to kill someone". This was most disquieting to the young man and far from encouraging him in further investigations along sinus lines; but thank God, he had the courage of his convictions and now is before you today, ready to stand or fall with those same implicit beliefs.

And now, Gentlemen, I am done. I appreciate this presentation has been rambling and often far afield from the province of laryngology, or for that matter even medicine, but its intention has been largely for the ears of our younger brethern. We must always look forward, not, as the Chinese do, backward with their ancestor worship, else we remain as they have, stationary for the last five thousand years, but with the spirit of progress guiding our thoughts, we can revert to the Chinese and their serenity of purpose, gleaning much from their phychology and when beset with discouragement and doubts, we can indeed say with our celestial friends, "Even tho the wolves may howl, the moon shines serenely on".

BULLER, THE OPHTHALMOLOGIST, POLITZER, THE OTOLOGIST, AND LEFFERTS, THE LARYNGOLOGIST.

HERBERT S. BIRKETT, C.B., M.D., LL.D. (McGILL)*

MONTREAL, CANADA.

Mr. President and Fellows:—

Allow me to express my deep appreciation of the honor conferred not only upon me, but also upon the medical profession of Canada and McGill University in particular, by inviting me to be the guest of honor of this important Association.

"Let us now praise famous men, and our fathers that begat us, ... their names shall remain forever and their glory shall not be blotted out." It is in the spirt of these everliving words from Ecclesiasticus, that we shall best understand the subject which I have chosen, namely, "Buller, the Ophthalmologist, Politzer, the Otologist, and Lefferts, the Laryngologist". The following are the reasons for my choice: First, the outstanding ability of these men in their respective specialities; second, my professional and personal association with each—Buller, from the privilege of being a student and then a house surgeon of his, and later from my close association of twenty years with him as a colleague and a friend; Politzer, from the good fortune of spending a year in his clinic now some forty years ago; and Lefferts, from the pleasure of attending his clinic from time to time. Further, I have selected these men as representing the high ideals which this Society stands for, and I shall consider each in the order corresponding to the title of this Society.

FRANK BULLER, THE OPHTHALMOLOGIST.

1844-1906.

Frank Buller was born in Campbellford, Canada, May 4th., 1844. His parents had emigrated from England to the United

*Professor of Oto-Laryngology, McGill University; Chief of the Department of Oto-Laryngology, Royal Victoria Hospital, Montreal.

States of America in 1831, and later resided in Cobourg, Canada. In 1856, the family returned to the United States and settled for a time in Iowa, where they experienced pioneer life.

Returning to Canada, Buller lived with his uncle, Judge Boucher, at Peterborough, where he attended the High School. Later, following the custom of the time, he became apprenticed to Dr. George Burnham, at the same time attending Rolfe's Medical School in Toronto, from which he graduated in 1868. After graduation, he spent one year in general practice in the State of Michigan.

In 1870, we find him working with von Graefe in Berlin. At the outbreak of the Franco-Prussian War, he became an Assistant Surgeon in the German Army, carrying on his duties as such until the end of the war, when he was appointed to the staff of the Graefe-Euer Hospital in Berlin. He studied pathology under Virchow and physics under von Helmholtz, thus placing his future work in ophthalmology upon a sound foundation.

Desirous of furthering his studies, he proceeded to London, where he became attached to the Royal Ophthalmic Hospital at Moorfields as a clinical assistant, and it was at this period that Buller introduced to London the direct method of ophthalmoscopy which he had learned in Germany. Whilst occupying this junior position, he passed his examinations as a Member of the Royal College of Surgeons, which enabled him to qualify as a candidate for the position of Junior House Surgeon at Moorfields. His application was successful, and later he was appointed Senior House Surgeon. Altogether he spent four years here, and during his term of service he devised the well known "Buller's eye shield". At this time, he met Dr. Francis J. Shepherd and the late Sir William Osler, both of whom persuaded him to settle in Montreal, which he did in 1876.

After six years, Buller was now highly qualified to enter the field of ophthalmology, having thus set the example, at least in Canada, of the necessity of long preparation, which in later years he strongly advocated to those seeking advice in the training of all specialities. He was the first in Montreal, and probably in Canada, to practice ophthalmology as a speciality, for until that time diseases of the eye had been treated by the general surgeon. As ophthalmology was still in its infancy and Canada only sparsely populated, it was

necessary to follow the custom existing on the North American continent of combining otology with ophthalmology. Buller was the first to be appointed in these specialities to the staff of the Montreal General Hospital, and as this was the teaching institution of the Medical Faculty of McGill University, he was appointed in 1883 to the newly created Chair, which he occupied until his death.

In appearance Buller, was tall and broad shouldered, with a well shaped head and square lower jaw, a slightly florid complexion and penetrating blue eyes. His manner at times seemed brusque and impatient, but it was only so to those who were slow to interpret his purpose; on all other occasions he was kind, considerate and generous. Self reliant by nature, and fortified by his thoro training, he worked with an assurance that engendered confidence, and the impression which remains is one of strength and decision yet innate refinement.

A striking characteristic was his interest in obscure cases, and the possibility of defeat called forth his greatest effort. This made a reputation which he was ever jealous to guard, and as an instance I well remember his operating upon a case of cataract in an extremely poor patient who positively refused to enter the hospital, and therefore the operation was done in the house amidst the most unhygienic surroundings. During the operation, a certain amount of vitreous escaped, necessitating constant cold applications, and as those were the days of Sairey Gamp, Buller, in order to insure a successful result, remained with the patient, changing the compresses all thru the night.

During my visits to the leading clinics in Great Britain and Europe, I have seen no one who surpassed him, not only in his operative technic, but also in his wonderful surgical instinct, which served him in great stead in his decisions as to the necessity of operating. He was very proud of the steadiness of his hand, and would frequently demonstrate it by holding a Graefe knife in each hand, so that when they were approached to each other, the points would touch. He was also ingenious, thoughtful and resourceful; for instance, he was the first to realize that the eyeball after an operation might become infected from secretions of the lacrimal sac, and when this was evident, he applied a temporary catgut ligature to each canaliculus. Again, he was the first to regard nasal conditions as a possible cause of certain ocular phenom-

ena. His ingenuity was ever ready to overcome difficulties, and this was well illustrated by a case of very marked epicanthus due to a saddle nose caused by an injury during childhood. This epicanthus was corrected by the implantation beneath the skin of a piece of solid glass, which had previously been moulded from carefully prepared casts. This, to my knowledge, is the first attempt at nasal prosthesis, and was performed thirty-five years ago; it stood the test of time for twenty years, when the patient was lost sight of. Buller's plastic work on the eyelids was marvelous, and I never saw a case in which the skin grafts failed to take.

His writings, seventy-six in number, extending over a period of thirty years, are work of a teacher and a practitioner, rather than the product of an original investigator, and may be classified under three heads:—

First, a group which marks him as the first exponent on this continent of the new ophthalmology of von Helmholtz, Donders and von Graefe.

Second, his clinical writings, which are no mere compilations, but were given out only after intimate study of personal experience and accompanied by illustrative cases. His last paper "Methyl Alcohol Blindness" was written while he was in failing health, and is described by de Schweinitz as "by far the most important contribution to the subject, and one to which too high praise cannot be given".

The third group of papers shows his remarkable ability and his means of overcoming difficult obstacles in the practice of ophthalmology as follows:—

1. The Buller eye shield.

2. Modification of Crichett's idea of slitting the outer canthus in gonorrheal ophthalmia, in order to apply strong nitrate of silver to the everted conjunctiva.

3. Modification of Mule's operation. Instead of suturing the conjunctiva and the sclera together, he did them separately, one vertically and the other horizontally, thus preventing the spread of possible infection from one layer to another.

4. The ligation of the canaliculi to prevent septic material passing from the lacrimal sac during operations on the globe.

5. A trial frame.

By these valuable and original contributions, the science of ophthalmology was greatly advanced. By his death, which occurred in October, 1906, from pernicious anemia, the scien-

tific world lost one of its pioneers, respected and beloved by all who knew him.

Adam Politzer, the Otologist.
1835-1920.

Adam Politzer was born in Alberti, Hungary, October 1st., 1835, of German-Jewish extraction, the son of an educated merchant, and the grandson of a physician. He studied at the University of Vienna, and in 1859 obtained the degrees of Doctor of Medicine and Master of Gynecology.

Upon advice, he took up the study of otology at once in the laboratory of Lüdwig, of Vienna, and while studying here, he published his first papers, one on "The Innervation of the Muscles of the Middle Ear" and a second one on "The Effect of Air Pressure in the Tympanic Cavity on Pressure in the Labyrinth." He then went to Würzburg, when under Kolliker he learned the histologic technic for examining the labyrinth. With Müller he studied physiology, and demonstrated "The Relation of the Trigeminus to the Eustachian Tube", and at the same time studied clinical otology with von Tröltsch. He then proceeded to Heidelberg where he studied acoustics under von Helmholtz. His next step was to London, where he studied pathologic anatomy with Toynbee, from whom he received the inspiration which resulted in his own marvelous collection of dissected specimens, which is second only to that of Toynbee. Politzer next went to Paris, where he studied physiology under Claude Bernard, acoustics under König, and clinical otology under Meniére. While here, he produced a paper on "The Vibration of the Drum and Ossicles of the Tympanum".

It is interesting to summarize his preparatory training before he began to practice as an aurist in Vienna in 1861. He had studied physiology with Lüdwig, Müller, and Claude Bernard; acoustics with von Helmholtz and König; histology with Kolliker; pathologic anatomy with Toynbee; and clinical otology with von Tröltsch, Toynbee and Meniére. What a splendid example he set to those wishing to specialize in otology, and how few of this generation have followed it!

Upon his return to Vienna, he established an otologic clinic, consisting of one small room in the clinic of Oppolzer, and having been made a Docent in the University of Vienna, he began teaching, and it is significant to note that in his

first class of four was Lucae, who later became the renowned professor of otology in Berlin.

Politzer, realizing the importance of studying the pathology of all ear conditions for the better understanding of symptoms and treatment, was given access to cases in the poorhouse, and it was from this source that he derived the material for his famous collection of specimens, sixty of which are in the Müttermuseum in Philadelphia and the remainder in the aural clinic in Vienna.

He had a talent for drawing and painting, which he gradually developed and put to great practical use in demonstrating aural conditions, not only in his clinic, but later in illustrating an atlas on the drum membrane, which was published in 1865. I well remember, when studying under him forty years ago, the value of the sketches which he made of each patient after examination and so fixed on the patient's shoulder, that one could readily compare the sketch with the ear under examination; this made his teaching additionally clear and instructive.

In 1869, Politzer, associated with Schwartze and von Tröltsch, founded the "Archiv für Ohrenheilkunde" of which he became Editor.

In 1870, Politzer was made Professor Extraordinarius, and owing to the rapid increase in the size of his clinic, two extra wards of ten beds each were added, and these were divided between men and women patients, Gruber having charge of the men and Politzer of the women. At that time, and even when I was studying there, all operations were done in a screened off portion, at one end of the same ward in which he taught.

His classical book on otology, which appeared in 1878, went thru five editions and was translated into several languages.

In 1887, his twenty-fifth anniversary as Professor of Otology was celebrated by his friend and pupils, who presented him with a portrait of himself and an address. In his reply, Politzer made some wise remarks regarding the speciality, the necessity of hard work and careful and truthful observation, etc.

In 1895, in conjunction with Gruber and Urbantschitsch, he founded the Otological Society of Austria, and Politzer's last

public appearance was upon the occasion of its twenty-fifth anniversary in 1920.

The following year, Politzer was made Professor Ordinarius, and upon the retirement of Gruber in 1897, he was made the sole director of the otologic clinic of the University of Vienna. In 1899, his clinic was enlarged by two additional rooms. In 1902, he was made Hofrat Professor.

In 1893, he came to America representing the Government of Austria at the Chicago Exhibition, and on his way there, he stopped in Montreal and did me the honor of calling upon me.

Politzer's last publication was "The History of Otology", the first volume of which appeared in 1907; the second volume was published after his retirement in 1913, to which I had the privilege of contributing the chapter on "The History of Otology in Canada".

When Politzer, in 1907, had reached the age of seventy-two years, he was retired according to the regulations of the University and made Emeritus Professor, and on this occasion he was presented by his friends with a handsome bronze relief, a replica of which I personally treasure.

Politzer had a happy disposition, a charming personality, sparkling, intellectual dark eyes and a musical voice. He was always neat in his appearance. He worked diligently but never hurriedly. He denounced everything wrong, but never made personal remarks. He was admired by his assistants and students, and ever showed a deep interest in their work and progress. He taught that to advance in science, one had to be truthful and realistic; diligence and endurance were essential. On one occasion he said that "genius is not able to accomplish lasting results without indefatigable work and diligence". He emphasized "continuous observance of clinical phenomena with autopsy findings and objective observation of the patient"; this shows his own method of approaching a problem.

He was an accomplished linquist, speaking German, English, French, Hungarian, Italian and Spanish fluently, and a little Russian.

He published over one hundred articles, covering every phase of otology, many of which are classics.

He was a connoisseur of art and a collector of minatures and lithographs, all of which he proudly showed to a few of his privileged students.

After his retirement in 1907, up to his death in 1920, he worked indefatigably all the time, and even in his eighty-fifth year he still performed difficult intratympanic operations. He retained his faculties and youthful energy until the end, and it is said that he was always ready to adopt anything new which was brought out.

The Great War affected him very much owing to his wide, international friendships. The coal shortage was so great, that he was forced to live in a boarding house for warmth, but visited his apartment and laboratory every day. He died suddenly of apoplexy in August, 1920, after practically sixty years' devotion to otology.

George Morewood Lefferts, the Laryngologist. 1846-1920.

George M. Lefferts was born in Brooklyn, New York, February 24th, 1846. His paternal grandfather was of Dutch extraction, and his maternal grandfather was of English extraction.

He graduated from the College of Physicians and Surgeons, Columbia University, in 1870, subsequently holding the position of a resident at both the Bellevue and St. Luke's Hospitals in New York. At the expiration of his term of service he went to Vienna, and there studied laryngology for two years under von Schrötter and Karl Störk. It was while here that he first met the late Sir Felix Semon, with whom he maintained a lifelong friendship. Upon his return to New York, he was appointed to the teaching staff of the College of Physicians and Surgeons in Laryngology, and in 1876 he was made Professor.

Lefferts' lectures were always carefully prepared, well delivered and most interestingly given. They were illustrated by specially prepared charts (500 in all) and further elaborated by blackboard drawings. These lectures were at the same time illustrated by cases carefully selected from his clinic. Practical work was carried out in small groups of six students each, thus ensuring more individual teaching.

Special laryngologic societies were first formed in the United States of America. The first one to be established was known as the New York Laryngological Society, founded October 13th, 1873, by Clinton Wagner, and one of its charter members was Lefferts. In 1878, at a meeting held in Buffalo,

a number of physicians who had identified themselves for some years past with laryngology, decided to form a National Association "for the advancement of the special department of surgery in which they were chiefly interested." As a result of this meeting, the American Laryngological Association was founded, of which Lefferts was the first Secretary.

The first journal devoted entirely to the speciality of laryngology was the "Archives of Laryngology," founded in 1880, of which Lefferts was one of the editors. This journal was a marvel of production, but unfortunately only lived to have four volumes published. An interesting and original method of reporting progress in this science was that instituted by Lefferts in 1874, by the publication of a series of abstracts from time to time in the New York Medical Journal. He was also a collaborator for the "Archives de Laryngologie" and the "Internationales Centralblatt für Laryngologie" (now called the "Internationales Centralblatt für Laryngologie, Rhinologie und verwandte Wissenschaften"). Lefferts was largely responsible for a complete bibliography of laryngology, in which work Morell Mackenzie and Elsberg were associated with him.

Lefferts was instrumental in establishing the New York Metropolitan Throat Hospital, one of the earliest of its kind in the United States.

His recognition of the eversion of the laryngeal ventricles, in 1876, is the first recorded in America, and he was also the first to operate upon a case successfully by laryngofissure with tracheotomy.

The study of the diseases of the nose, which up to the year 1874 had been given but scant attention, received a great impulse from the work of Lefferts and others.

In the United States of America, postgraduate teaching was first instituted in the city of New York, in 1877, chiefly owing to the initiative of Lefferts and others associated with him, who established regularly organized instruction in the Demilt Dispensary, the most important dispensary at that time.

In early life, Lefferts developed hip joint disease, which left him lame, but this affliction served only to stimulate his ambition. Altho he was a man of ample wealth and social position, he never allowed either to lead him from his chosen profession. His unusual grace and talent were devoted un-

sparingly to his life work and were reflected in his distinguished and handsome countenance. As Delavan says of him, "Bright and genial in conversation, he was ever a welcomed companion and charming host." One of his chief characteristics was his love of order, and his watchword was preparedness.

After an active practice of thirty-seven years, Lefferts retired to his country home, where he spent ten peaceful years, succumbing to an attack of cardiac angina, September 21st, 1920.

May I conclude my remarks upon these illustrious men with the following words of James Russell Lowell:

"The longer on this earth we live
And weigh the various qualities of men,
The more we feel the high, stern featured beauty
Of plain devotedness to duty.
Steadfast and still, nor paid with mortal praise,
But finding ample recompense
For life's ungarland expense
In work done squarely and unwasted days."

I wish to express my indebtedness to Dr. Archibald Malloch, Librarian of the New York Academy of Medicine, and to Dr. W. J. McNally, of Montreal, for their valuable assistance in the research work of this paper; also to the writers concerned in these references, for much information from whose articles I have freely abstracted.

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THE ETIOLOGY OF FOCAL INFECTION—MEDICAL ASPECTS.*

RUSSELL L. HADEN, M.D.

KANSAS CITY, MISSOURI.

The systemic expression of a focal infection is the result of the interaction of focus and patient. Here, as in most other infections, the activity of the infecting bacteria and the resisting powers of the infected individual are of prime importance. A discussion of the medical aspects of the etiology of focal infection must necessarily be limited largely to consideration of these two factors.

Before these factors are discussed in greater detail, one point demands the greatest emphasis. This is the need for a general medical survey of every patient presenting evidence suggestive of systemic disease of focal origin. Such a survey necessarily includes a carefully taken history, a competent physical examination, and such laboratory procedures as are indicated. In this way, the possibility of systemic disease being related to a focus of infection can usually be determined, and the relative importance of the different factors be evaluated. The internist is constantly seeing patients who have suffered at the hands of specialists for the lack of such survey. Often haphazard therapeutic procedures are undertaken without a clear perception of the problem in mind. Two patients seen very recently illustrate the truth of these statements. One patient had had a number of teeth removed for pain in the back. No relief was obtained, since the pain was due to a stone in the kidney. Another had all the teeth extracted for weakness. Again no relief followed, since the symptoms were due to an unrecognized pernicious anemia, which probably has little relation to focal infection. The observing internist in such a survey keeps in mind the ever present possibility that symptoms commonly ascribed to focal infection may be due to very different causes. A toxemia of intestinal origin

*University of Kansas School of Medicine, Kansas City, Kansas.

and other metabolic derangements may produce disease clinically identical with that characteristic of focal infection. A differentiation is often difficult to make.

Every careful student of focal infection is impressed with the fact that no subject in medicine must be handled with more mature clinical judgment, when he sees the frequent examples of the harmful results of the reckless removal of foci, especially the teeth, the frequent ascribing of conditions to focal infection when due to entirely different causes, and the careless promises for the relief of systemic disease by the removal of foci. The more thoroly one studies the subject, the more cautious one becomes. This does not alter the conclusion that focal infection is of the greatest importance in the causation of systemic disease.

Having determined that a patient's symptoms are probably due to a focal infection, the primary factors of focus and individual must be considered and evaluated. The first problem is to find the focus. Often this is apparent. In other cases it is very difficult to find. The focus may be located in many different places in the body. Theoretically, a focus may exist at any point in the body in which a localized area of infection may persist under pressure. Cases are reported in which a chronic localized infection around a toenail or in some other obscure site has resulted in serious metastatic disease. From a practical standpoint, only the teeth, the tonsils, the sinuses, the gastrointestinal tract, the prostrate and seminal vesicles, and the female generative organs need be considered. This seems to be a large field, which may, however, be still further delimited by the statement that the last three named are relatively infrequent sites of foci of clinical importance in systemic disease.

In every individual suffering from systemic focal disease, however, all possibilities must be evaluated. The probability of each possible site will vary with the individual studied. Certain general principles may aid in determining the focus. In young individuals, metastatic disease arises very uncommonly from the gastrointestinal and genitourinary tracts. At this age period, the infection is most apt to arise from lymphoid tissue, hence the tonsils are most frequently at fault. In older individuals, the other possibilities must be considered, altho here also the prostate, seminal vesicles and female gen-

erative organs are of minor importance, considering the field as a whole.

The tonsils may be a focus at any age and should never be eliminated as a possible focus on the basis of negative clinical findings. Infection in sealed off cypts occurs very commonly. In the presence of manifest systemic disease of focal origin, a tonsillectomy is always indicated if the tonsils are not already removed, provided there are no contraindications for operation. The same may be said for tonsillar remnants, which are frequently encountered after the tonsils have been removed. Infection in sinuses may occur at any age interval and give rise to metastatic disease.

Considering all cases of metastatic disease of focal origin, the teeth certainly hold first place as a source of infection. Infected teeth are almost universal, due to the widespread occurrence of dental caries and the practice in the past of killing pulps. Dental infection is especially responsible for systemic disease of focal origin occurring after middle age, and for the diseases resulting from continuous damage over a long period of time, such as myocarditis. In a large group of individuals of all ages in Kansas City, I found 92% had one or more pulpless teeth. An average of 1.4 abscessed teeth per person was found.

The development of systemic disease from a focus seems dependent in large measure on the conditions under which the bacteria live in the focus. This fact must determine the acquirement of pathogenicity, the conditions in bone being especially favorable for the development of pathogenic properties. It is probable, that in most instances at the time bacteria first enter a focus, they have little pathogenicity. Living in tissues deficient in oxygen aids materially the development of specific toxins. Rosenow showed many years ago that the toxins of the pneumococcus, to which the streptococci of focal infection are closely related, develop more readily in the absence of oxygen. In experimental work, specific localizing properties can be preserved only under relatively anaerobic conditions.

Physician and dentist agree that teeth showing radiographic evidence of infection should be removed. There has been much difference of opinion concerning the disposition of pulpless teeth which do not show radiographic findings characteristic of infection. Bacteriologic studies have shown that

there is little difference in the frequency of infection in the two groups. If the removal of all possible foci is indicated, no pulpless teeth should be allowed to remain in the mouth.

In trying, in a given case, to determine in the presence of several possible foci which one is probably the most important factor, some information may be gained from knowing the type of systemic disease present. Thus acute rheumatic fever, chorea, and rheumatic endocarditis and myocarditis arise probably entirely in lymph-adenoid tissue. Other types of endocarditis or myocarditis may come from any focus. Acute glomerulonephritis is most apt to come from the tonsils. Other types of kidney disease, such as pyelonephritis may come from other foci. Chronic infectious arthritis, on the other hand, comes by far more frequently from dental infection than from other sources.

A very important factor in focal infection is the multiplicity of foci. One constantly sees patients who present a number of possible foci. It may be proven by animal experimentation, that even in the presence of multiple foci, only one may be causing the systemic trouble. This can be determined only experimentally, however. Often it is difficult to determine the order in which foci should be removed. This will depend in part on the systemic manifestations. It is unwise to proceed too rapidly. In deciding precedence of removal between tonsils and infected teeth, usually the dental infection should be removed first. The only safe rule is to remove all possible foci if practicable.

The other aspect of the question of etiologic factors is the reaction of the host to the invading organisms. An individual may harbor one or more foci of infection without ever developing demonstrable systemic disease from the focus or foci. Another person with only a single focus, seemingly much less important, may develop serious disease. The great difference in susceptibility is impossible to foresee and difficult to evaluate. It is probably constantly varying in every individual. Among the factors influencing it, are the presence of chronic debilitating disease, such as nephritis, and the occurrence of an acute infection, such as measles or influenza. Age is also a most important factor. Especially after middle age, is the resistance to infection on the wane. Even with a fall in resistance, however, a patient cannot develop systemic disease of focal origin unless a focus be present. I often use this simile

in explaining to patients the importance of removal of foci. Suppose two individuals are exposed simultaneously to diphtheria. One is susceptible to the disease and contracts the infection; the other is immune and does not develop diphtheria. Here, the determining factor is the degree of resistance, yet the susceptible individual would not develop the disease if he had not gotten the diphtheria bacillus in his throat. In the same way, a person may develop arthritis due to low resistance, yet the important factor is a focus, without which the disease of focal origin could not develop. Often little can be done to influence the resistance. Much can be done in eliminating foci.

The correct recognition of the factor of resistance in focal infection materially influences the prognosis. Given two patients with focal systemic disease of equal intensity, a better clinical result is to be expected from the removal of a virulent infection in an individual with good resistance than from the removal of a mild infection in an individual with low resistance. Two patients suffering from arthritis seen at the same time, recently, illustrate well this point. One was a young man on crutches, with a severe subacute arthritis following a marked alveolar infection. The other was an elderly lady, who had developed an arthritis following the shock of a fall in which an arm was broken. She, too, had every indication that the focus of the joint trouble was in dental infection. In the first patient, the removal of the infected teeth enabled the patient to lay aside his crutches the next day. An excellent result was to be expected. In the second patient, the removal of the teeth had little effect on the source of the arthritis. Little was to be expected.

Foci of infection should not be disturbed unless there is something definite to be gained from the operation, either in preventing further disease or in aiding in the cure of systemic disease of focal origin. The patient's physical condition may contraindicate any operative procedure. The systemic disease may be all out of proportion to the local lesion. In other patients, the structural changes may be beyond repair, and of so serious a nature as to make the local lesion a minimal part of the clinical picture. Here, it is often unwise to stir up foci even when causally related to systemic disease. Caution should be exercised in recommending the removal of foci in elderly individuals unless manifest advantage is to be gained thereby.

One more point is, it seems to me, of paramount importance. The recognition of the role of chronic foci or infection in systemic disease has been a gradual one. It has been brought about by the clinical study of the end results of such localized infections and by animal experimentation. We are now continually seeing patients suffering from systemic disease of focal origin and attempting to cure them by removal of the focus or foci. So often the curative attempts are most disappointing. The symptoms of the systemic disease often represent the effects of the bacterial poison over a period of years and are dependent upon organic changes in tissue which cannot be undone. The realization of these facts serves only to emphasize the early removal of foci of infection. We should rather try to prevent the development of systemic disease by removing foci, than attempt to cure systemic disease by this procedure. Especially with the increasing interest in periodic health examinations, more opportunities will be given for the early detection of foci and for giving the proper advice concerning them.

I may summarize what I have attempted to discuss in a few words.

The first requisite in the management of focal infection is a sane general survey of the patient, keeping in mind the possibility that the symptoms often ascribed to focal infection may be due to entirely different causes.

In the vast majority of cases, the foci are found in the teeth, tonsils and sinuses.

In determining the disposition of foci, factors influencing the resistance of the individual must be evaluated, and the structural damage already done must be considered.

Very often the systemic disease is dependent upon continuous damage over a long period of time. Eradicating the foci may avail little.

Where the removal of foci is indicated, all possible foci must be removed.

The experimental and clinical study of focal infection emphasizes the important relation of foci to systemic disease, and the need for the early recognition and removal of foci before systemic disease has resulted.

The systemic disease should be cured before it begins.

FOCAL INFECTION AND ELECTIVE LOCALIZATION IN THE PATHOGENESIS OF DISEASES OF THE EYE.*

EDWARD C. ROSENOW, M.D.

ROCHESTER, MINNESOTA.

In no field, perhaps, is there more convincing clinical evidence of the causal relationship of focal infections to disease in remote parts of the body than in that of diseases of the eye. The importance of acute septic infections as a cause of metastatic disease of the eye and other organs has long been recognized. The idea, however, that mild, often symptomless, localized infections, such as occur so commonly about teeth and in tonsils, sinuses, or prostate, may also predispose to diseases of the eye and other remote structure, while recognized by a few observers for a long time, gained little headway until the idea was placed on a sound basis about fourteen years ago by Billings. He first emphasized its importance on the basis especially of a coordinated clinical and experimental study of cases. Since then, great progress have been made. The idea is applicable to such a wide range of diseases, and the mild localized infections occur commonly in so many structures, that the quite general application of the principles involved has greatly modified the practice of medicine and surgery, including the various specialties so well represented in the American Academy of Ophthalmology and Oto-Laryngology, and even the practice of dentistry.

In a large number of papers, the importance of this factor as a cause of various diseases of the eye has been emphasized thru the report of cases in which foci were demonstrable, and relief attended removal of foci of infection. These and other corroborative facts leave no doubt of the correctness of the idea. Much still remains to be learned concerning the proportion of cases in which this factor is directly responsible, and concerning the relative importance of the site of the focus or

*Division of Experimental Bacteriology, The Mayo Foundation.

foci. However, from a study of the many reports already available, it appears that while localized infections in tonsils, sinuses and intestinal and genitourinary tracts are of great importance, especially since they may cause infection of teeth, those in and about the teeth appear especially prone to cause ocular manifestations.

While there is quite general agreement regarding the etiologic relationship of focal infection wherever found to diseases of the eye, there is still a difference of opinion regarding their nature, and whether they are due to localization of organisms or their toxins, especially in those that run a chronic course and that manifest periodic exacerbations and remissions. This is particularly true in those believed to be due to streptococci, because many clinicians still have the impression that all members of this group of organisms are highly virulent, and that any disease in the eye due to localization of streptococci must needs be severe and associated with suppuration. This is erroneous, for it has been proved beyond doubt that in the streptococcal group, more, perhaps, than in any other species of bacteria, there are individual strains of almost every degree of virulence. There are strains that affect, in diverse manner, a wide range of tissues or organs, while others have specific or elective affinity for particular structures without noticeably affecting others. Clinical evidence in support of these experimental findings is not lacking, for in some of the diseases of the eye, such as "rheumatic" iritis, the ocular symptoms are only a part of similar manifestations in joints, muscles, heart and other structures, while in most of them the condition in the eye, so far as can be determined, is the only manifestation of disease. The pathogenesis of this group, especially the mild, yet menacing affections, is still a puzzling problem. The difficulties of solution are many. Relief from symptoms following removal of foci of infection, while good evidence of causal relationship, still leaves open the question whether the lesions in the eye were the result of localization of bacteria or their toxins only. Material for cultural and microscopic study are not obtainable and, judging by experimental findings, even if the fluid in the anterior chamber were aspirated, it would likely be sterile when living organisms were present in the affected tissues. Systemic reactions are slight, cultures from the blood are negative, antibody production is minimal, and hence the usual

agglutination reactions with suspected organisms are of little or no diagnostic value. Antisyphilitic therapeutic tests and the tuberculin test cannot always be relied on to distinguish syphilitic and tuberculous from other forms of infection of the eye.

I wish now to consider the experimental results of the intravenous injection of bacteria from foci of infection, which throw more light, perhaps, than other methods of study on the mechanism and cause of nontuberculous and nonsyphilitic metastatic infections of the eye.

In connection with a long series of experiments in which animals were injected under uniform conditions with streptococci isolated from patients manifesting different systemic diseases, I have had the opportunity to study the lesions of the eye that sometimes developed in the injected animals. Unilateral panophthalmia was noted five times, twice following injection of the streptococcus isolated from the joints in cases of rheumatic fever, once with the streptococcus isolated from herpes zoster after passage thru one animal, and once after the injection of *Streptococcus mucosus* isolated from the pus in the eyeball in a case of metastatic ophthalmia following tonsillitis and otitis media. Hemorrhages, with or without episcleritis, were noted in the region of the limbus in fourteen instances. Iritis or iridocyclitis developed in nine, four times after injection of certain laboratory strains, three times with streptococci from rheumatic fever, and twice following injection of streptococci from small pus pockets in tonsils of normal persons. Conjunctivitis, associated with the streptococcus injected, occurred in ten instances, and ulcer of the cornea in one. Hemorrhages in the posterior part of the eyeball were not often looked for, but were found in two instances. The lesions of the eye were acute and consisted of edema and extravasation of erythrocytes, associated with a variable degree of infiltration by leucocytes. Thrombosis of small vessels rarely occurred. The organism injected was demonstrated in the lesions in large or small numbers by cultural methods and microscopically in sections. This was true in some instances after the animal had seemingly recovered and cultures from the blood proved negative. The bacteria were found within blood vessels adjoining the lesions, and free or within leucocytes in the areas of hemorrhage and infiltration. Suppurative iritis was found only once. The frequent occurrence of

lesions in the ciliary body, iris and limbus in these animals parallels, roughly, what occurs in man. This might be attributed to the fact that here, as in the structure about joints and tendinous ends of muscles, there is a gradation from an abundant to a scanty blood supply, and hence a gradation in the supply of available oxygen, thus inviting localization and affording favorable conditions for growth of bacteria. This circumstance would also favor the production of lesions by bacterial or other toxins.

It must not be supposed that the occurrence of the lesions in the eye was wholly accidental, for they occurred more often with strains isolated from diseases in which affections of the eye are relatively common, such as in rheumatic fever and herpes zoster, than with strains isolated from diseases such as appendicitis, ulcer of stomach and cholecystitis, in which lesions of the eye are rare. The production of ophthalmia with the streptococcus isolated from the eye in cases of metastatic ophthalmia, is also suggestive. These experimental results and many clinical facts indicate that localization may be dependent on inherent qualities of the various streptococci injected. Precise evidence in favor of this view was obtained some years ago in connection with experiments on the transmutation of pneumococci and streptococci. In this work, various strains of pneumococci and streptococci of low virulence were passed successively thru animals by intravenous injection, in order to raise their virulence, to note the changes in localizing power, if any, and to observe changes in cultural features and in morphologic characteristics. Thus, a strain which had been isolated eleven years previously as a pneumococcus, and which had long since lost its virulence, was passed successively thru seventeen animals without producing ocular lesions in a single instance; but, in the eighteenth passage, it produced suppurative conjunctivitis in the only rabbit injected. In the nineteenth passage, it produced iritis (rabbit). In the twentieth passage, it produced suppurative conjunctivitis associated with iritis and marked hemorrhages and infiltration at the limbus in a dog, and marked conjunctival and scleral hemorrhages, especially at the limbus, in a rabbit. Subsequent passages failed to produce ocular lesions. Similar results were obtained from injection of two strains of pneumococci in successive animals. At the time, these grew as streptococci, and had lost their

virulence by long cultivation on artificial media; each produced iritis in the tenth and fourteenth passage thru an animal, while all the other injections failed to produce lesions of the eye. The injection of a strain which originally had been isolated as a hemolytic streptococcus from "milk slime," but which had lost its hemolytic property and had acquired the properties of a typical Streptococcus viridans by growth in symbiosis with Bacillus subtilis on a blood-agar plate, produced no lesions in the first seven injections, but in the eighth passage thru animals produced hemorrhages about the limbus and in the retina in two rabbits injected; in all subsequent passages no ocular lesions developed.

The results of these animal experiments, in which marked changes in localizing power in different structures were noted, furnished the experimental basis for the theory of elective localization and led to an extended study, under a constant technic, of the localizing power of bacteria isolated from foci of infection and systemic lesions in diseases affecting widely different structures and of widely different character. Striking evidence of specific or elective localizing power, corresponding in situation to the organs or structures involved in the patient, was obtained in many of these, including diseases of the eye. The importance of strict attention to technical details, such as the proper selection of cases, use of partial tension cultures and the injection of freshly isolated cultures, became apparent and has since been repeatedly emphasized by myself and by Haden.

In 1916, Irons, Brown and Nadler reported the successful production of iridocyclitis in rabbits by the intravenous injection of streptococci isolated in cases of acute iridocyclitis. They were able to show that the power of the streptococcus to invade special tissues may change within a short time during residence in the host, during passage thru an animal, and in cultures.

Numerous attempts have been made, with only partial success, to find a method that might be relied on to preserve elective localizing power and viability for long periods. Preservation of latent life under reduced oxygen tension in tall tubes of meat-mash infusions, or in shake cultures containing relatively few colonies in glucose-brain or ascites-glucose agar, in dense suspension in gycerine (two parts) and saturated sodium chlorid solution (one part), have often

served to preserve viability and elective localizing and other specific properties for a long time. In exceptional instances, this was true for as long as eight years, when the corresponding strains, grown aerobically, had lost elective localizing power during several daily transfers. Subcultures rapidly made (from four to eight each twenty-four hours) in previously warmed mediums which afford a gradient of oxygen tension, such as glucose-brain broth, often serve to maintain specific localizing power and agglutinating properties for many generations. Elective localizing power of streptococci was preserved often for many weeks or months within the pulp chamber or periapical tissues of artificially devitalized and infected teeth in dogs.

The use of proper cultural methods is not the only requirement for the successful application of the principles involved in the study of elective localization, especially as pertains to localized inflammatory processes in different diseases. Here difficult procedures and judgment of elusive factors are often necessary. We have learned that a mere correct diagnosis may not be sufficient. The condition at the time of study should also be considered. A higher incidence of localizations and more marked lesions in animals are obtained if cultures are secured at the time of acute systemic manifestations, or during exacerbations of symptoms in chronic conditions, than during quiescent intervals. Great care is exercised in the collection of material for study, both as regards the clinical observations and the prevention of contamination by extraneous organisms. Pus from the depths of tonsils is expressed, and cultures made from this instead of swabbings from the surface or from single crypts. The urethra is irrigated with sterile solution of sodium chlorid before the prostate is massaged to obtain material for cultures. Material from the nasopharynx is obtained by swabbings high behind the palate, with cotton swabs on aluminum wire bent to a suitable angle, without touching the tongue. Pus from the depths of pyorrhea pockets or discharging sinuses is aspirated into fine pipettes, after the surface has been cleansed with alcohol or other antiseptic. Pulpless teeth are extracted under as nearly sterile conditions as possible, and, in special instances, the apical end is resected for culture before the tooth is extracted. Culture controls are made of the mucous membrane before and after sterilization, and of the overlying bone after the mucoperiosteal flap is resected.

An opportunity to determine whether streptococci from foci of infection in diseases of the eye possess elective localizing power, and hence produce lesions in the eye, was afforded in April, 1919, in a case of recurring iritis, which was reported by Benedict. The clinical findings and results of the cultures and animal experiments are representative of others studied since by myself, by Benedict, and by my associates. The patient, a young woman, was seen during the early stages of an attack of iritis similar, in every respect, to others which had occurred previously. The general examination was negative. Radiographs of the teeth revealed four impacted, unerupted molars, and fracture of the roots of both upper central incisors, sustained by a fall eleven years before. The impacted and fractured teeth were extracted and the sockets of the incisors curetted. Recovery from the iritis was rapid. Smears from the foul smelling pus found in the enlarged pulp chamber of one incisor showed gram postive diplococci and gram negative fusiform bacilli. Aerobic cultures remained sterile, while partial tension cultures in tall tubes of glucose-blood broth yielded an anaerobic streptococcus which produced iritis in a series of rabbits. Cultures from the turbid fluid from the anterior chamber remained sterile, while those from emulsions of the rabbit's iris yielded the organism injected; this again produced iritis on intravenous inoculation. In several of the rabbits, hemorrhages had also occurred into the dental nerves and pulps, and into muscles. The streptococcus was demonstrated microscopically in the lesions in the eye and proved absent in the unaffected tissues. Thus, by the use of partial tension cultures, it was possible to isolate a streptococcus with which the disease of the patient was reproduced; if aerobic cultures only were made, as is still so often the case, the teeth would have been pronounced sterile.

At about the same time, a similar partial tension streptococcus, which produced lesions of the eye and of the pulps and nerves of teeth, was isolated from an inflamed dental pulp in the tooth of a man who had suffered from recurring attacks of rheumatic pains in the back, arms, and shoulders and thru the head.

Lewis during the same year produced iritis in a rabbit by injecting a culture of Streptococcus viridans isolated from a periapical infection in a patient suffering from acute iritis.

Meisser and I have since found a high incidence of elective localization in the eyes of rabbits injected intravenously with steptococci from dental and tonsillar infections in patients suffering from acute iritis, which was only rarely obtained with streptococci from chronic cases of iritis or other chronic affections of the eye.

In 1921, I reported the results of a study of two cases in which the eyesight had been well-nigh lost from recurrent intraocular hemorrhages, and in which focal infection had not previously been suspected as a causative factor. The method of intravenous injection of cultures into animals directly from foci of infection yielded a striking illustration of the mechanism of the occurrence of this most deplorable of ocular conditions.

The cultures for injection in one were obtained from the vital pulp of a badly decayed tooth; in the other, from a suppurating sinus surrounding a third molar. Both yielded positive results. Of the nine rabbits injected, lesions of the eye developed in six. In one, panophthalmia with hemorrhages in the retina resulted. In the others, swollen, whitish areas and hemorrhages in the retina were demonstrated with the ophthalmoscope or by microscopic examination of sections. Streptococci were demonstrated in the lesions. Similar experiments made in other cases at the same time produced no lesions of the eyes. Clinical evidence of a direct connection between focus and ocular condition (in this instance probably due to absorption of toxin) was present in the case in which the suppurating sinus was found in the jaw. The degree of vision fluctuated, in a remarkable manner, with the stoppage or drainage of the pus exuding from the tooth. With lack of drainage, even for a day, the vision diminished on repeated occasions, and increased when drainage was again established.

Benedict, 1921, reported the clinical and experimental results in a series of cases, indicating that the ocular lesions attributable to focal infections, especially various forms of iritis, are associated with localization and growth of organisms in the lesions and are not due to toxins.

Haden, in 1923, in a long series of experiments in cases of acute iritis, episcleritis, uveitis, and hyalitis, has had similar results. He reported finding lesions of the eye in 68 per cent of sixty-six rabbits injected with freshly isolated cultures from teeth of patients suffering from metastatic infections of the

eye. Of the control group, in which parallel injections were made of cultures from infected teeth in persons that were well or were suffering from some other condition, lesions of the eye developed in only 14.8 per cent of 169 rabbits injected. He also emphasized the importance of the use of proper technic in order to avoid misleading results, and among other conclusions, stated that "teeth which are negative in the radiograph may bear as important an etiologic relation to disease as those which show evident infection." The ocular lesions induced in the animals resembled, in many respects, those noted in patients, as regards clinical appearance, structures involved and microscopic changes, and were due in all instances to localization of the organism.

Nickel has reported the results of an extensive study (under a constant technic) of the localizing power of bacteria from foci of infection in tonsils, teeth, prostrate and cervix, in patients suffering from various diseases, including diseases of the eye. The factor of specificity or elective localizing power was brought out in a striking manner in each of six groups of cases studied. Moreover, he found what I had repeatedly observed, namely, that the best results are obtained in cases of acute disease or during periods of exacerbation in cases of chronic disease. His results from a routine study in which all cases were included, and in cases selected according to the type of focus at hand and acuteness of symptoms, as well as in control cases, are strikingly brought out in the tabulation. The high incidence (75 per cent) of animals that showed lesions of the eye in the selected cases is especially noteworthy; such lesions were found in only 34 per cent in the routine cases and in 1 per cent of the control group. In this connection the fact should be mentioned that he has found that lesions of the eye, especially congestion of vessels of the iris and circumcorneal region, have no doubt been missed by others; such congestion sometimes occurs and disappears within from six to twelve hours after intravenous injection, and may not progress to exudation and hemorrhage. Moreover, in a series of as yet unpublished experiments, he has been able to show that cultures of streptococci having elective affinity for the eye produce a filterable toxin that affects specifically the eyes of rabbits when injected intravenously. Control experiments with filtrates of streptococci having no affinity for the eye have little or no effect. I have obtained

similar results with filtrates of cultures of a bacillus and diplococcus isolated from the eyes of horses affected with periodic ophthalmia.

COMPARISON OF RESULTS IN ANIMALS INJECTED WITH CULTURES FROM ROUTINE AND SELECTED CASES. (NICKEL.)

	Selected Cases	Animals Injected	Positive Animals	Percentage of Animals Showing Specific Localization: Selected Cases	Control Cases	Routine Cases
Arthritis	82	220	173	77	7	51
Myocarditis	9	29	18	62	3	39
Myositis	10	23	16	69	4	38
Lesions of the eye	11	24	18	75	1	34
Lesions of the skin	4	8	4	50	1	36
Peptic ulcer	39	116	80	68	3	40

Benedict, von Lackum and Nickel have shown the importance of streptococcal infections in the prostate and cervix in relation to diseases of the eyes. They have reported clinical results in a series of cases and the results of the injection of animals with freshly isolated cultures. Of twenty-four cases studied experimentally, fourteen yielded positive results in animals. Lesions of the eye developed in 29.5 per cent of sixty-one rabbits injected. Excluding cases in which the condition of the eye was chronic, the focus questionable and the symptoms quiescent, lesions in the eye were produced in 67 per cent of the animals injected. This was in sharp contrast to the incidence of 7 per cent of 181 animals injected with seventy-nine strains obtained from the teeth, tonsils, prostate, and cervix of patients whose conditions were not definitely diagnosed, and who presented no ocular symptoms. The authors found that the causative organism isolated from these foci was a slightly hemolytic or green producing streptococcus, requiring partial tension methods for its isolation, and that local treatment and autogenous vaccines made from the strains obtained from experimental lesions of the eye in rabbits often greatly benefited the patient.

The underlying physiochemical conditions that determine elective localization of living bacteria are undoubtedly similar to, yet as obscure as, those that determine the specific pharmacologic action of drugs and chemicals. However, the fact that certain streptococci possessing elective localizing power, especially those from the lesions in ulcer of the stomach, myositis, epidemic hiccough and encephalitis, and now from iritis and other ocular infections, have been shown to produce poisons or

toxins free in broth cultures and within the bacterial cell, which have the power to injure electively the tissues or organs in which the respective living bacteria localize and produce lesions, throws a clearer light on how the bacteria overcome the inherent resistance of tissues against invasion.

Summary and Conclusions.

The more important clinical and experimental studies on the pathogenesis of nonsyphilitic and nontuberculous intrinsic infections of the eye, and the requirements for the successful application of the methods of study are reported. The method of intravenous injection of primary (often mixed) or freshly isolated, pure cultures of material from foci of infection has led to a better understanding of how these seemingly harmless localized areas of infection, often small and in obscure places, cause ocular manifestations; and by this method the causative organisms usually responsible have frequently been isolated.

In the light of the newer knowledge, foci of infection, wherever found, should be looked on as a place where favorable conditions are afforded for entrance into the blood or lymph stream of bacteria and their toxic products, and where they may acquire or maintain peculiar or relatively high invasive power. They make for a forced relationship between parasite and host.

The good effects commonly noted following removal of foci of infection support the experimental findings, justify a thoro consideration of their existence and call for removal or cure, as far as possible, of focal infection in every obscure clinical case. The successful application of the methods of study, while simple, require unusual facility of cooperation between bacteriologist and clinician. The experimental results indicate clearly that those lesions in the eye which are associated with exudation, even tho slight, are usually due to actual localization of microorganisms, while the milder manifestations may sometimes be due to absorption of toxins which are formed in the focus or elsewhere and reach the eye in the blood stream.

Localization of the bacteria in the eye may sometimes be accidental and a part of other disease manifestations. However, the animal experiments, now amply corroborated, indicate clearly that localization and growth in most instances are due to peculiar, acquired or inherent, properties within the

bacteria themselves, and to their power to localize electively, and that this is in part due to the production of a toxin or poison which affects, specifically, the tissues in which localization and growth occur.

Among the microorganisms isolated which manifested the greatest elective localizing power, and with which the common forms of diseases of the eye have been reproduced, is a streptococcus which usually produces greenish or slightly hemolytic colonies on blood-agar, which requires a reduced oxygen tension for its isolation, and from which autogenous therapeutic vaccines, of great value in many cases, have been prepared.

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OTOLARYNGOLOGIC PHASE OF FOCAL INFECTIONS.

H. W. Lyman, M.D.*

ST. LOUIS, MISSOURI.

In a symposium of this character, it is essential that each participant confine his remarks as closely as possible to the portion of the subject assigned to him. I, therefore, will endeavor to limit myself to the ear, nose, and throat phase of this fascinating subject.

My experience has been chiefly clinical, and it is from that point of view that I present these observations.

The numerous articles on this subject attest the widespread interest of the profession in the relation of focal infections to general diseases. The work of Billings and Rosenow stands out prominently in placing the subject upon a scientific basis, because of their careful clinical observations, confirmed by closely checked bacteriologic studies, and extensive animal experimentation.

So many of these foci of infection are in the ear, nose and throat, that this subject is of the greatest importance to the otolaryngologist.

Keiper[1] lists nearly seventy diseases in which the tonsils have been considered the offending focus, and this list by no means includes all of the diseases which other writers have attributed to the same cause.

Statements such as this may well make us pause and ask whether we are not being carried by our enthusiasm beyond the limits of prudence.

Speaking of the clinical phase of focal infections, D. J. McCarthy[2] (of the University of Pennsylvania School of Medicine) says:

"In studying the influence of focal infections, we should fully realize that we are dealing largely with theoretic considerations and not with a definite demonstrable fact, or series

*Assistant Professor Clinical Otolaryngology, Washington University, School of Medicine.

of facts, subject to a testing process such as the pathologist or bacteriologist gives out in a laboratory experiment. Any data on this subject, necessarily, must be largely a matter of impression on the part of the investigator, tinged somewhat by his state of mind toward the problem under consideration. In the vast majority of cases, the focal infectious process acts on an already existing condition of undernutrition, anemia, endocrin imbalance, etc.,—more especially in individuals with arterial hypotension than those with normal blood pressure or arterial hypertension."

The work of Dr. Amy Daniels, at the University of Iowa, who is able to produce, at will, infection in the paranasal sinuses of rats, by a diet deficient in fat soluble vitamin A, while the control animals remain unaffected, calls attention to the importance of nutrition in this question of focal infections.

These observations emphasize the fact that this problem involves not merely the discovery and removal of a focus of infection, but the study of a diseased patient, requiring the closest coperation between the otolaryngologist, the internist, the dentist, and the bacteriologist, and the closer this cooperation, the better will be the result for our patients, and the more exact will be our knowledge of the subject now under discussion.

These general factors also probably account for the fact that many of us harbor foci of infection without developing any of the diseases commonly attributed to this cause, and that frequently such diseases undergo resolution without the removal of any offending focus, while in other cases the removal of all discoverable foci fails to result in clinical improvement.

Methods of Infection: Apart from direct extension to surrounding tissues, infections in the upper respiratory tract may invade other structures by the direct inhalation or swallowing of the infective organism, by the entrance of toxins or the organism itself into the blood stream, or into the lymphatic system.

Tonsils: That infected tonsils may be the cause of general diseases is so universally accepted, that their removal, either as a curative or prophylactic measure, requires no defense. The question of what constitutes a diseased tonsil is one that the laryngologist must decide for himself in each

individual case. The author's opinion is, that if there is a history of previous infection of the tonsils, or clinical evidence, such as induration of the tonsil tissue, redness of the anterior pillars, or expressible pus, the patient will be benefited by their removal, especially if consultation with the internist discloses any of the general diseases in which a focus of infection may be considered a factor.

ADENOIDS: That the adenoids may harbor infectious pus, which is not discoverable by the use of the ordinary postnasal mirror, is easily demonstrated by the use of Kelley's direct vision adenotome. This instrument frequently enables one to observe pus being squeezed out of apparently simple hypertrophied adenoids, during their removal.

SINUSES: The discovery of foci of infection in the paranasal sinuses is often quite simple. A mere inspection of the nasal cavity may disclose the presence of pus, and the source from which it comes is determinable with comparative ease.

In other cases, however, the most painstaking care in repeated examinations, with the use of every available aid, such as transillumination, X-ray plates taken from different angles, the use of lipiodol by displacement instillation as described by Proetz, and even the exploration of the sinuses themselves, may be necessary before the question of an infective focus can be decided. In children, sinus infection is quite apt to be overlooked, and the use of the X-ray and irrigation of the antra will frequently show an infection which routine inspection would not disclose.

The discovery of an infection in the paranasal sinuses does not necessarily indicate radical surgery, as many of these cases will clear up under conservative treatment, and this should be tried if the general condition of the patient will permit. In children, especially, the removal of tonsils and adenoids, and the use of ephedrin in the nose is frequently all that is necessary. If this does not suffice, the antra may be opened below the lower turbinate. This is often the only intranasal surgery indicated, it being seldom that operations on the ethmoids or sphenoids are necessary.

While not a typical example of focal infection, as generally considered, sphenoiditis, either purulent or hyperplastic, may cause irritation of the nasal ganglion, with the manifold symptoms described by Sluder. The injection of this ganglion with five per cent phenol in alcohol may relieve these symptoms.

but the removal of the underlying cause, namely, the infection in the sphenoid sinus, seems the logical method of attack in these cases.

THE EAR: Infections in the ear have not been as generally recognized as foci causing general diseases, as have infections in the teeth, tonsils, and sinuses.

Our experience in the St. Louis Children's Hospital has convinced us that infections in the middle ear and mastoid antrum in infants may be the cause of gastrointestinal disturbances, commonly described as "Cholera Infantum". As the development of this phase of focal infections is recent, it will be entered into in some detail.

Hartmann,[3] in 1898, called attention to the fact that infants with infections in the ear often suffered from diarrhea, vomiting, and loss of weight, and that these symptoms improved after paracentesis.

Maurice Renaud,[4] of Paris, in 1921, however, seems to have been the first to have recognized the fact that this infection may involve the mastoid antrum, and that incision into the tympanic cavity is not sufficient to relieve the condition. After autopsies which had disclosed pus in the mastoid antra of seventy infants who had died of so called "Cholera Infantum," he opened the mastoid antra of ten such infants suffering from diarrhea, vomiting, and loss of weight. All of these showed improvement of the gastrointestinal symptoms, but nine eventully died, and only one survived. This high mortality, he believed, was due to the fact that the operation was not performed until the infants were in extremis, and that had he operated earlier, the results would have been better.

In the fall of 1923, autopsies at the St. Louis Children's Hospital disclosed pus in the mastoid antra of infants who had died of gastrointestinal disturbances not due to food poisoning but to some parenteral infection, and in whom no other focus of infection was found.

At this time we did not know of the work of Renaud, or that of Dean and Byfield at the University of Iowa. This work was carried on in close cooperation with the Department of Pediatrics, under the direction of Dr. W. McKim Marriott.

In May, 1925, Dr. A. M. Alden and the writer presented a paper based on mastoid operations in fifteen cases of infants under two years of age, suffering from diarrhea, vomit-

ing, and loss of weight due to parenteral infections, with eight recoveries and seven deaths.

Ten of the twelve cultures in these cases showed hemolytic streptococcus growths, and it seemed that there must be some particular strain of this organism which had a special affinity for the gastrointestinal tract, but our subsequent experience did not confirm this assumption.

During the winter of 1926-27, mastoid operations were performed on forty-three infants under two years of age, thirty of whom presented marked gastrointestinal symptoms. Twenty-two of these thirty recovered, and eight died.

Cultures were obtained from sixty-seven mastoids in this series of forty-three cases. Seventy-nine growths were obtained, and seventeen different organisms were isolated. These different organisms showed no special relationship to the gastrointestinal symptoms, all of the more common varieties occurring both in cases with and without gastrointestinal symptoms. So that while the hemolytic streptococcus may be a special factor in the production of gastrointestinal symptoms in infants, other organisms also produce the same effect. These infants fail to gain normally in weight and suffer more or less from gastrointestinal disturbances, and in the more severe cases have marked diarrhea, vomiting, loss of weight, toxicity, and a septic temperature. These symptoms are not affected by any change of diet, but, after the mastoid antra are opened, the infants thrive on the same diet which they could not assimilate before the operation.

The ear, besides acting as a focus of infection, is frequently affected by foci in other parts of the body, and this should be carefully considered in the study and treatment of all cases of deafness of the nonsuppurative type, whether of the conducting mechanism or the perceiving mechanism type, as it is generally accepted that a chronic, low grade infectious process is frequently the underlying cause, and, unless this is discovered and removed, we cannot hope to check the progress of the disease.

Vertigo denotes a disturbance of the vestibular apparatus, and, when actual disease of the labyrinth or central nervous system is eliminated, it is usually due to a toxic irritation from some infected focus. While this focus may be in the teeth, appendix, gall bladder, female pelvic organs, or prostate, it is not infrequently in the upper respiratory tract. A careful

use of the vestibular tests will enable us to distinguish these cases of toxic labyrinthitis from those due to organic lesions in the labyrinth and brain, the characteristic features in these toxic cases being that all of the reactions, both to turning and caloric tests, are proportionately affected. In the early or irritative stage, they are all exaggerated, and we speak of the labyrinths as being "hypersensitive." In the later stages, when actual destruction of the labyrinthine structures occurs, these reactions are proportionately diminished; and if total destruction of the labyrinth occurs, the reactions are abolished.

That toxemia from oronasal sepsis may accompany neurasthenia or mental unsoundness, is common knowledge. Psychic disturbances in focal sepsis are striking and often disastrous.

Among the most prominent classes of disease which our experience has caused us to attribute to focal infection, are the rheumatic and arthritic cases. While some of the older and more serious types do not show remarkable improvement, the general results in these cases are such as to convince us that foci of infection are an etiologic factor, and that our lack of success can be attributed to a failure to eliminate all foci, or to the existence of some other overlooked factor.

In cardiac cases, both muscular and valvular, we are convinced of the value of the elimination of foci of infection, both as a preventative and a curative measure. In these cases, close cooperation with the internist is most essential, as they are bad operative risks and the greatest care must be exercised to have the patient in the best possible condition when any surgical work is attempted, this, together with the post-operative care, being much more important than the mere operative procedure.

In the genitourinary group: Cases of pyelitis respond as a matter of routine to the elimination of infective foci.

Hemorrhagic or glomerular nephritis is influenced most favorably by the removal of foci of infection in the throat, nose, and ear.

I wish to cite briefly the cases of two brothers which illustrate this:

C. L., aged five years, entered the hospital February 24, 1927, with acute hemorrhagic nephritis, following an acute tonsillitis. The X-ray showed a cloudiness of both antra. March 5th, irrigation of both antra disclosed pus. Windows into the antra were made beneath the lower turbinates and

one-fourth per cent mercurochrome instilled. March 9th, the tonsils and adenoids were removed. The patient was discharged March 12th. The urine was free from blood, albumin and casts, and has remained so up to the present time.

His brother, J. L., aged four years, developed acute tonsillitis March 15th, probably contracted by kissing Charles on his return from the hospital. On March 24th, he developed hemorrhagic nephritis with increased temperature, albumin and blood in the urine. He was sent into the hospital March 31st for removal of tonsils and adenoids, but, because of the acuteness of his condition, operation was postponed until April 29th. The operation was uneventful and the child has continued free from nephritis.

Cases of nephrosis or parenchymatous nephritis, with edema, present a less favorable picture. They are very poor operative risks and show a marked tendency to relapse. Whether this is due to the type of organism, or to some other factor, is a question requiring further research for its solution.

In the ocular group of diseases commonly attributed to focal infections, the results are usually most favorable, and sometimes almost magical, as, for example, in the case of a boy of eighteen, referred to me, who was almost totally blind in one eye. An infected sphenoid and ethmoid region was exenterated, with almost immediate restoration of sight, and he amused himself the same afternoon by watching a baseball game from the hospital window, using only the previously blind eye. When cases are referred to us by an oculist, we should be most careful and thoro in our search for a focus of infection, and be willing to operate on slighter evidence of infection than we would if they were uncomplicated otolaryngologic cases.

Pulmonary Group: Tuberculosis will only be mentioned to call attention to the possibility of infection by way of the lymphatics from the upper air passages to the apex and hilum of the lung, but there is a type of case in children which is often diagnosed as turberculosis, especially by the X-ray, which we believe to be a nontubercular infection from the upper air passages. The citation of the following case will briefly illustrate this type:

E. H., aged 7 years, admitted January 19, 1924, with a history of anemia, undernourishment, sore throat, bronchitis, and pus in the urine. A tentative diagnosis of tuberculosis had

also been made. Weight 39 pounds, Height 42¼ inches. The X-ray report of the chest was as follows:

(a) X-ray of chest, anteroposterior: "Much increase of hilus shadow on either side. Lung markings are thickened, nodular, and extend fairly well out. Thruout the upper section on either side there is a large amount of fine soft and coarse parenchymatous mottling. Diagnosis: Pulmonary Tuberculosis."

(b) X-ray of sinuses: "There is a general haze over both antra." Diagnosis: Bilateral Maxillary Sinusitis." The tuberculin skin reaction was negative. On admission he had a bilateral acute otitis media, which promptly subsided after paracentesis.

Nose and throat examination revealed a tonsillar stump remaining on the left side, and a mass of adenoid tissue in the nasopharynx. The nasal mucous membrane was swollen and covered with a thin mucoid secretion. The remains of the tonsils and adenoids were removed. Windows were made in the antra, below the lower turbinates, and one-fourth per cent mercurochrome instilled.

The nasal infection cleared up, the appetite improved, and the weight increased.

Patient was seen February 2, 1925. Weight 52½ pounds; height 46½ inches. Urine, negative. Ears, nose, and throat negative.

Asthma and Hay Fever: Altho there is neither time nor occasion to discuss this complicated question in detail, many cases are relieved or improved by the elimination of foci of infection in the upper air passages.

Glandular system: The direct connection of the lymphatics of the upper air passages with the deep and superficial cervical glands accounts for most of the infections of these structures. In fact, enlargement or induration of these glands should cause us to search most diligently for a focus of infection either in the throat or the sinuses.

Infectious diseases, such as scarlet fever, diphtheria, measles, poliomyelitis, etc., will only be mentioned to call attention to the upper air passages acting as a port of entry for the general infection.

In diseases of the nervous system, we feel that chorea may be benefited by the removal of infected tonsils, and the correction of other foci in the upper passages.

Septicemia, with the port of entry in the ear, nose, or throat, is much more frequent, especially in children, than is realized, unless routine blood cultures are taken. While many of these cases are unrecognized and undergo resolution spontaneously, the removal of the offending focus is the surest method of curing them.

Skin lesions: Under this heading the writer desires to call attention to a condition in infants which seems to have a direct relation to infected tonsils and adenoids, namely, erythredema, sometimes called acrodynia or "Pink disease." This affection usually occurs in children from three months to three years of age. The onset is insidious, with poor appetite, malnutrition, restlessness, irritability, wakefulness, profuse sweating, and a leucocyte count of ten thousand to fifteen thousand. The characteristic feature, however, is the eruption, which is macular in type with miliary papules. This may involve the entire body, but is most marked in the extremities. The hands and feet are red and swollen, but feel cold to the touch. Desquamation, ulceration, and loss of the nails, or even the toes, may occur. The nervous symptoms are especially prominent. The persistent insomnia, the restlessness, irritability, and constant rubbing or scratching of the extremities are most striking. The duration is given as months or even years.

We have recently seen several of these cases, and their improvement after the removal of tonsils and adenoids has been much more prompt than the usual description would lead one to expect.

One of the most striking features of our experience with diseases due to focal infections, has been that the results obtained are by far the most favorable if the offending organism is the hemolytic streptococcus.

Conclusions.

1. Focal infections are frequently the cause of general diseases.

2. A large proportion of these foci are found in the ear, nose, and throat.

3. The fact that multiple infections may exist must be borne in mind and all possible foci considered.

4. Finally the writer again wishes to emphasize the statement that this problem does not consist merely in the dis-

covery and elimination of an offending focus, but is one which requires the most careful study of the case from every angle, and the closest cooperation between the various consultants.

5. While this general care and observation is essential in these cases, the prime importance of discovering and correcting any focus of infection remains, and we can safely state our belief that no patient is made worse by the elimination of foci of infection in the ear, nose, and throat.

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THE OPHTHALMOLOGIC PHASE OF FOCAL INFECTION.

RICHARD J. TIVNEN, M.D., LL.D., F.A.C.S.

CHICAGO, ILLINOIS.

In my consideration of the ophthalmologic phase of focal infection, I purpose to discuss the subject in a more or less general way. I will refrain, therefore, from presenting case histories or clinical experiences or dwelling upon any special pathologic entity.

As a preliminary to the consideration of the specific topic assigned me, it may be of interest to refer briefly to some principles involved in the focal infection concept. It appears that we are indebted principally to two men, Billings and Rosenow, for establishing on a scientific basis the focal infection concept as an etiologic factor in morbid processes. Billings' article on focal infection, published in 1912, was an illuminating contribution to clinical medicine. This contribution is generally regarded as being the first systematized clinical presentation which definitely established the focal infection concept as a factor in the production of pathologic processes. Rosenow's research work on the elective localization of streptococci, published in 1915, was likewise an epoch making experimental contribution to the subject,—a mile-post in the progress of modern medicine.

Billings defines a focal infection as "A metastatic systemic or local condition due to infectious microorganisms or their toxins carried in the blood or the lymph stream from a focus or foci of infection" and a focus of infection as "A localized or circumscribed area of tissue containing pathogenic microorganisms." It is generally conceded that certain factors predispose an individual to such infections. Practically all such factors are, in the main, embodied in the principle of lowered resistance—a locus minoris, or a reduction of the individual's general defensive equipment.

The following mode of dissemination of bacteria and toxic products from the focus of infection is given by Billings:

1. Hematogenous: The microorganisms of the focus of infection may readily reach the blood stream thru the surrounding capillaries. Bacteriemia is doubtless a very common occurrence. Bacteriemia may be likened to multiple small emboli carried in the blood stream.

2. Lymphogenous: The infectious agents in a focus may pass to more distant tissues thru the lymph stream.

3. Systemic Intoxication: The bacilli of diphtheria and tetanus excrete soluble toxins in the focus of infection which are absorbable and cause systemic intoxication. Most other pathogenic bacteria are endotoxic, and the poisonous material is released by autolysis of the dead microorganisms.

4. Foci of Infection and Anaphylaxis: Foci of infection may be the sites of the parenteral introduction of a foreign protein and the cause of anaphylactic sensitization of the body tissues. This foreign protein may be the bacterial protein of the invader, or the protein elaborated in the focus of infection by the reaction between the microorganism and the local infected tissues."

The outstanding conclusions one reaches in an investigation of this subject are: first, that the doctrine of focal infection is an accepted scientific fact; second, that seldom, perhaps never, does a local lesion, a focus of infection, remain purely local. Sooner or later the focus becomes active and in consequence the organism reacts either as a whole or in part, producing either a general systemic infection or a local infection in a single structure, like the eye or cranial cavity. A familiar example of this is lymph node infection, which, as we know, may confer protection on the individual by serving for a time as a "detention station" for infection, but is likely at some future time to develop activity and itself become a source of infection.

From the standpoint of the ophthalmologist, the foci of infection which in the literature receive most frequent mention are the tonsils, the teeth, the oral cavity, the nasal accessory sinuses, the nose, pharynx, the prostate, the deep urethra, and the rectum. This list is, of course, not submitted as a complete one. The consensus of opinion appears to be that the teeth rank first as the most frequent offender. The diseases of the eye which it is assumed may develop from these various sources of focal infection are exceedingly numerous. Emerson, in 1917, presented the following list: "Retrobulbar neu-

ritis from the infection in the ethmoids and sphenoids; acute edema of the lids and congestion of the conjunctiva from the ethmoids; pain and photophobia from acute congestion in the anterior ethmoid region; iritis from a focus in the tonsil, teeth, or sinuses; blocking and infection of the tear duct from the obstruction of the nasal end of duct; orbital cellulitis from the ethmoids; optic atrophy from the pressure of tumors in the ethmoid region; ocular displacement and diplopia from pressure in the ethmoid region; and supposed connection of phlyctenular keratitis with infected tonsils and adenoids." One may add to this list; tuberculosis, syphilis, gonorrhea, sympathetic ophthalmia and endogenous infections. In the ten years that has passed since Emerson presented this list, the subject has received a very considerable amount of study. As a result, many of the ocular difficulties he records have received confirmation, some still remain in the column of speculation, while certain others have been added to the list.

The Nasal Accessory Sinuses: In striving for an anatomic basis, as an explanation of ocular pathology, the nasal accessory sinuses have borne the emphasis of investigators. On the anatomic side, the particular sinuses which have enlisted the greatest interest in this connection, are the posterior ethmoid cell and sphenoid sinus, in their relation to the optic nerve, the optic canal and chiasm. The salient anatomic features of these corelated structures may be summarized briefly as follows: the orbit, the globe, and a considerable part of the orbital portion of the optic nerve are practically surrounded by the nasal sinuses. White's conclusions based on an anatomic X-ray study of the optic canal, are as follows: "The normal optic canal is 5.5 mm. in diameter; it varies from 3.5 to 6.5 mm.; the smaller the canal, the more the danger of permanent loss of vision and the greater the necessity for operation." Loeb's conclusions, based on the study of 15 heads are: "The optic chiasm is usually in relation with one or both sphenoid sinuses; in no instance in these heads, with the ethmoids; in five instances (one third), one sphenoid is in relation with both optic nerves, the other sphenoid not participating; as a rule, the last posterior ethmoidal cell (sometimes there are two) has a very slight relation with the optic nerve at the posterior external angle just at the roof." Crane describes the vascular connections and drainage of this region as follows: "The nervehead is supplied and drained by a

network of arteries and veins which form an integral part of the sinus circulation; the intracanalicular section of the nerve is supplied by small muscular branches of the ophthalmic artery and is drained by the vein of Vossius, which in turn drains into the cavernous sinus; numerous veins from the periosteum of the posterior orbit and the periosteum of the posterior ethmoidal cell and sphenoid sinus flow into the vein of Vossius; the lymphatic circulation between the posterior sinuses and the posterior orbital structures form a network of communication." Onodi and Loeb have shown that the partition between the optic canal and the sphenoid and posterior ethmoid cell is not infrequently thin, and occasionally exhibits dehiscences; the sheath of the optic nerve and the periosteum of the canal are also intimately connected; the canalis opticus incloses the nerve in a cavity with unyielding bony walls, and is therefore vulnerable to pressure. Posey states, "That owing to the fact that the nerves which supply the extraocular muscles are in close relationship with the sinuses, they may be paralyzed by pressure or by toxic influences resulting from sinus inflammation." Gradle believes that, "An extension of a pathologic process from the sinuses must of necessity be along the venous or lymph channels, and if the advance be not stopped by free anterior nasal drainage, will eventually involve the periosteum of the orbit, the dura-periosteum of the canal and probably the optic nerve. Such involvement is probably in the nature of a perivascular or perilymphatic edema, and as such causes pressure upon the nerve; the manifestation of such pressure depends upon the location of the pressure and may be enlargement of the blind spot (pressure upon the periphery of the optic nerve), central scotoma (pressure from the central vein upon the papillomacular bundle), ring scotoma, or complete amaurosis (diffuse pressure thruout the entire nerve)."

Hajek, Skillern, Onodi, Schaeffer, and practically every other observer, are unanimous in the opinion that ocular infection dependent upon a focus of infection in the nasal accessory sinuses is relatively not of frequent occurrence. Irons and Brown, in a study of 200 cases to determine the etiology of iritis, covering a period of approximately seven years, record but 3 cases of the nasal accessory sinuses being the cause of iritis.

White, as quoted by Ballenger, divides the pathology of loss of vision from sinus disease into four parts: "(1) Direct extension, (2) toxemia, (3) bacteriemia, and (4) hyperplasia. By direct extension, acute inflammatory infection in the posterior sinuses may extend rapidly, by continuity of tissue, to the sheath or to the optic nerve itself. Toxemia may play an important part; toxins originating in the sinuses or elsewhere may involve the nerve, while in bacteriemia or focal infection, microorganisms may be carried in the blood stream or lymph channels from the focus to the regions about the optic nerve. Hyperplasia of the sinuses in itself is not a direct factor, but as a predisposing factor, it is of considerable importance, as shown by the large number of eye lesions resulting from hyperplastic sinusitis." Ballenger notes the size of the middle and superior turbinates as of importance: "These turbinal bodies may become large and hyperplastic or cellular, thus causing faulty aeration and drainage of the posterior ethmoidal cells and sphenoid, thereby acting as important predisposing factors."

Skillern records Kuhnt's classification of the orbital complications of nasal accessory sinus disease as follows:

1st, Inflammatory: (a) affections of the conjunctiva and cornea; (b) affections of the uveal tract; (c) affections of the retina and optic nerve.

2nd, Functional: (a) muscular asthenopia; (b) loss of accommodation.

3rd, Mechanical: (a) disturbance of bulbar mobility; (b) irritation of the optic nerve thru pressure.

Disturbances of vision, he states, practically always results from posterior ethmoid and sphenoid disease. Hajek states that, "According to most statistics, the orbital complications are induced by the various accessory sinuses in the following order of frequency: first, the frontal sinus; second, the ethmoidal labyrinth; third, the maxillary antrum; fourth, the sphenoidal sinus."

The Teeth: There is a unanimity of agreement among observers that the teeth are perhaps the most frequent foci of ocular lesions. Benedict believes that, "Carefully observed cases of periostitis, orbital abscess, iritis and uveitis that can be ascribed to dental sepsis have been recognized and described." He presents the following possible modes of transmission of infection: "(1) Direct extension thru bone. The

tooth socket becomes the seat of an infection that destroys the bone of the upper jaws, and thru that directly affecting the orbital walls, finally produces an orbital cellulitis or retrobulbar neuritis; (2) Direct extension of the process along the periosteum of the malar bone, invading the orbit and giving rise to processes similar to those mentioned; (3) Transfer of organisms from the focus at the tooth to the eye by the blood stream. This is the most frequent means of transfer of infection from the teeth to the eye, and consequently is the most important." Fox states that "Dental affections provoke ocular troubles in two different ways: (1) by inflammation or irritation of the trigeminal nerve due to dental affection, causing reflex troubles in a manner similar to that in which neuralgia of the fifth nerve or tic douloureux is produced; (2) by the extension of an inflammatory process of the dental root toward the maxillary sinus, and thence toward the orbit by continuity and contiguity of the structure." Franchere records: "among the direct affects of apical abscesses and infective processes in the structures adjacent are photophobia, lacrimation, persistent conjunctival hyperemia, iritis, cyclitis, scleritis, and retinal hemorrhage." Lang, in a study of oral sepsis (quoted by Knapp), records that: "Out of 10,000 private cases, 215 cases of eye inflammation were due to sepsis, while 168 were attributed to other causes. Of the 215, 139 were due to pyorrhea. Of the 215 septic cases, the iris was affected 87 times, the ciliary body, 79, the choroid, 68, the retina, 28, the sclerotic, 20, the lens, 14, the cornea, 12 times, and there were 3 cases of detachment of the retina and 4 of optic neuritis or atrophy." Benedict notes that, "The iris and choroid are more often attacked by organisms from apical infection, altho optic neuritis, scleritis, keratitis and conjunctivitis are reported to have been found."

The Tonsils: The literature abounds with case reports of ocular disorders chargeable to tonsillar infection. Rosenow writes, "The results detailed in this ("Elective Localization of Streptococci") and in previous papers, seem to bring the necessary experimental proof that chronic foci of infection play a most important role in causing systemic disease. A focus, such as a pocket in the tonsil, which cannot heal for mechanical reasons and which is constantly filled with pus and necrotic material teeming with bacteria, must be recorded in the light of these findings as a culture tube, with a perme-

able wall affording abundant opportunity for the entrance of bacteria and their products." Ballenger notes, "Practically all writers agree that various pathogenic organisms gain an entrance to the deeper tissue of the tonsils, the lymphatic glands, the lungs, the heart, the kidneys and indeed, to the whole system thru these organs." Brown and Irons observe, "The tonsillar infections appear to be as important an etiologic factor in iritis as in arthritis and other metastatic lesions." Fuchs refers to tonsillar infections as being a possible etiologic factor in iritis, cyclitis, keratitis, bulbar neuritis, congestive optic neuritis, choroiditis, insufficiency of accommodation, and retinal hemorrhages. Knapp observes, "As the tonsils become infected in a number of infections, it is perfectly logical to expect toxic inflammations of the eye to follow, such as optic neuritis and paralysis of the ocular muscles. Mention should be made of the possibility of the infection extending along the intimate venous anastomoses of the pterygoid plexus, and causing a thrombophlebitis of the orbital veins and of the cavernous sinus, with the symptoms of an orbital abscess and blindness, or of an exophthalmos and death."

Clinical Manifestations.

The limitations of my paper preclude an extensive or detailed review of the literature of the special eye disorders attributed to focal infection. It is possible, therefore, to present only a more or less general outline of this phase of the subject, based on my investigation of the literature and my own personal experience, and to discuss in detail only a very few ocular lesions.

Iritis: Benedict from an investigation titled "The Character of Iritis Caused by Focal Infection" reaches the conclusion that, "Iritis is produced by bacteria arising from foci of infection around the roots of teeth and in the tonsil" * * * * * * "It is an inflammatory reaction brought about by the presence of bacteria in the tissue of the iris, carried to the iris by the blood stream from the foci of infection, and that such bacteria have the ability to grow, and do grow, in such invironments." He also reports the case of a man who suffered from recurring attacks of iritis during a period of six years; the attacks were always preceded by soreness of a right upper bicuspid, which came on about three days before the eye became inflamed; physical findings otherwise, negative; the

tooth referred to gave no roentgenologic evidence of apical infection, and an electric test indicated that the pulp was vital; the tooth was extracted and culture taken from the pulp, which, when injected into the blood stream of a rabbit, produced a hemorrhagic iritis of both eyes within a few hours; a subculture from this strain of organism produced a pericorneal injection within four hours; this entirely disappeared within twelve hours. Rosenow records that, "Iritis and certain other lesions of the eye generally thought to be toxic may be infectious in nature." He further states; "The frequent occurrence of lesions about the ciliary body or iris needs special emphasis. This might thought to be due to the fact that here, as in the structure about the joints and the more tendinous portion of muscles, there is a gradation from an abundant to a very scanty blood supply, and hence a gradation of the supply of available oxygen, thus inviting localization and affording opportunity for the growth of bacteria, a circumstance, which for the same reasons, would make for lesions by circulatory, toxic substances, no matter from what source." In his experimental work, he produced iritis in an animal by the injection of an organism from the tooth of a patient afflicted with muscular rheumatism. Fuchs states, "In many cases, iritis originates in a focus of purulent infection. Such focal infection may have its primary seat in the genitourinary tract, being then usually due to the gonococcus (gonorrheal iritis), sometimes to the colon bacillus. In other cases, it is derived from the teeth (especially pockets at roots of dead teeth), or less often from the accessory sinuses. In these cases, it is due usually to the streptococcus viridans (streptococcic iritis), less often to other germs (staphylococcus, influenza bacillus). Irons and Brown, in their classic investigation of the "Etiology of Iritis," based on a study of 200 cases record the following as among their findings: Tonsillar infections, 35 cases; dental, 27 cases; sinuses, 4 cases; tuberculosis, 8 cases; gonoccoccal, 10 cases; syphilis, approximately 38 cases. Kuhnt, referred to by Hajek, believes, "That even if the supposition of the direct dependence of an iritis upon an accessory sinus disease is unwarranted, there is no doubt, however, that the empyema acts as a predisposing factor in the disease of the uveal tract, and therefore the treatment of the accessory sinus has an unmistakable influence upon the course and upon the fundamental healing of the

diseases of the uveal tract, the optic nerve and the retina." Bulson in an investigation of the "Etiology and Treatment of Endogenous Iritis," based on the study of 100 cases, seen in private practice, presents the following analysis: "Syphilis, 33, with questionably two more credited to teeth and two more credited to tonsils; teeth, 32, including two associated with syphilis, and three associated with tonsils; tonsils, 22 and questionably three more associated with teeth; gonorrhea, 5; antrum, 3; frontal sinus, 1; sympathetic inflammation, 1; melanotic sarcoma, 1; undetermined, 2. My own experiences embrace a considerable number of cases of inflammatory lesions of the uveal tract, notably of the recurring type, which failed to respond to the usual management until various infective processes of the teeth, tonsil or sinuses, were discovered and corrected.

Phlyctenular Conjunctivitis: While the etiology of this disease is not definitely established, I believe that it is a manifestation of tuberculosis. In 1911, I made a study of 50 cases, and the result of this study impressed this belief strongly upon my mind. I mention the disease in connection with focal infection, first, because of my belief in its tubercular origin, and second, because of the well known clinical observation that infected tonsils and adenoids occur in a large percent of these cases, and the marked improvement which the patients exhibit after these diseased foci are removed would seem to justify the inclusion of phlyctenular conjunctivitis as a possible expression of focal infection.

Optic Neuritis, Retrobulbar Neuritis, Optic Atrophy: Considerable difference of opinion exists as to whether these pathologic processes are directly traceable to focal infection The following observations taken from Fuchs are pertinent: "Among other causes of neuritis, occasionally observed, is great chilling of the body; possibly here the real cause is a sinus infection." And again he states "Acute retrobulbar neuritis is either the result of inflammation of the surrounding orbital tissue or there is an idiopathic inflammation of the nerve. Catarrh of the nasal cavity passes to the accessory sinuses of the nose, especially to the ethmoid cells, and thence the inflammation is transmitted directly to the orbital tissues and to the optic nerve. Much more rarely, acute retrobulbar neuritis occurs in myelitis, in diseases of the tonsils or teeth. In not a few cases, disease of the optic nerve and uveitis may

be produced not by pressure, but by absorption of toxic matter from a chronically inflamed sinus. Chronic disease of the sinuses (especially of the sphenoidal sinus) may produce inflammation or atrophy of the optic nerve." Hajek states: "Among the diseases of the optic nerve accompanying affections of the accessory sinuses, retrobulbar neuritis plays an important part." Bordley's view is: "Assuming that the operator is qualified, and assuming also that all other probable causes have been eliminated, and that every means of diagnosis has been resorted to, I will suggest that when you are face to face with a serious optic nerve disturbance, the part of conservatism and good judgment requires an operative exploration of the ethmoidal and sphenoidal cells. I feel quite confident that visual disturbances are frequently the first suggestion of serious sinus disease, which may eventually lead to blindness or to death." de Schweinitz, in discussing Dr. Bordley's paper, stated, "I heartily concur in his contention, that in the presence of serious optic nerve disease, not otherwise perfectly explained, even if the nose shows no sign and the Roentgen ray gives no certain information, it is the business of the surgeon to explore." Schaeffer believes, "that it is established, that disease of sinuses may lead to an optic neuritis even to blindness." Skillern, states that, "Retrobulbar neuritis which may result in atrophy and loss of vision also occurs as a complication of accessory sinus disease." Ellett notes, "The ocular lesion in disturbance from posterior ethmoidal and sphenoidal disease is practically always of the optic nerve, which is certainly not often the case in affections of the anterior sinuses."

Choked Disc: Some confusion apparently still exists among observers in the use of the terms "papilledema" and "papillitis," the former, it is assumed, implying "edema," the latter "inflammation" of the nervehead. Cushing emphasizes this distinction: "It is perfectly conceivable that suppurative processes in the ethmoid cells adjacent to the optic foramina may set up similar disturbances in the optic nerves. But what is inconceivable, is that these processes should produce choked disc. It is important to bear in mind, that what by common usage is referred to as choked disc (Stauungspapilla, papilledema) is a condition brought about by mechanical causes, and that optic neuritis, papillitis and other terms are misnomers for this condition. Inflammatory processes may affect the

optic nerve in such a way as to produce reddening, injection and possibly such a degree of hyperemia and vascularity of the nervehead as to resemble the early hyperemic stage of a choked disc, but it is unbelievable that an infection of the accessory nasal sinuses, in the absence of increased intracranial tension, should produce an actual choked disc." Tooker observes that, "Varying opinions are held by equally competent observers as to the production of a choked disc or a papillitis of high degree by an infection in the nasal accessory sinuses." Horrax, in a study of cases, concludes: "Altho there are a few conditions other than brain tumor which may cause papilledema or a condition of the optic nerve simulating it, the percentage of instances in which it occurs in these conditions is extremely small as compared to tumor." White believes that, "Swollen discs from accessory sinuses, teeth, or tonsils is an unusual condition, is transitory, and is always associated with sudden and marked impairment of vision. Papilledema is the usual condition in brain tumors, and before advocating exenteration of the accessory sinuses, a very careful study of the neurologic side of the case should be made." He directs special attention to cases which exhibit loss of vision from intracranial neoplasms, and who also have definite nasal pathology, and observes: "The removal of the infection within the nose or throat naturally does not benefit the vision of patients with multiple sclerosis or acoustic neuromas." Bordley states: "It has been my privilege to see nasal sinus disease complicated by choking of the disc, but in every instance where the cranial contents were viewed either before or after death, there existed unmistakable evidence of an intracranial pressure." Sluder's opinion is: "Neurologists, for the most part, think that choked disc can be produced only by intracranial pressure, and that it is edema of the disc produced by cerebrospinal fluid pressed out in the sheath of the optic nerve. Ophthalmologists recognize what is the same lesion, that certainly arises from other causes also. * * * * * Many times I have had eye cases referred to me with the statement that there is a lesion, usually termed choked disc of from one to eight diopters, and no neurologic or other cause can be found for it. I have been able to recognize in these cases hyperplastic sphenoiditis, often with acute processes added, and have relieved the eye trouble by the post. ethmoidalsphenoidal operation." Elschnig (quoted by Knapp)

"Found that the ophthalmoscopic changes in optic nerve involvement secondary to sinus disease varies from a perfectly normal fundus to pronounced choked disc." Parker, in an experimental study on the mechanism of papilledema in nine dogs and three monkeys, first reduced the tension of one eye by sclerocorneal trephine operation, and afterward raised the intracranial pressure. "In every instance, the swelling of the disc appeared first in the eye showing the lesser tension." He concludes: "The results of the experimental work seem to confirm the mechanical theory." Eagleton is of the opinion that, "The generally accepted view of cerebral compression as the sole cause in their (optic neuritis and papilledema) production, is not consistent with clinical and experimental evidence." To those who may be interested, I recommend reference to the contributions of Cushing, Sluder, Tooker, Horrax, White, Bordley, Knapp, Parker, and Eggleton on this interesting subject.

Corneal Ulcer: de Schweinitz states: "The frequent relation of carious teeth and pyorrhea alveolaris to corneal ulceration is well established, and the irritation of a new dentition in young children has been found to be the cause of abscess or ulcer of the cornea. Some corneal ulcers appear to have been caused by diseases of the accessory sinuses, especially the ethmoid sinuses, and by purulent rhinitis, and if the tonsils are infected, they should be removed." There are frequent case reports in the literature which connect focal infection as an etiologic factor with corneal ulcer. Under the head of "Traumatic Lesions," I have referred to a personal experience.

Recurring Retinal Hemorrhages. This condition is rather frequently mentioned in the literature as being attributed to focal infection. F. Park Lewis expresses the opinion, that in such cases, "What actually occurs is the absorption into the blood of a soluble protein poison, usually given off from a focal infection, which has a solvent effect on the intercellular cement substance of the cell wall, and that the hemorrhage is not due to alteration of the blood pressure but to local lysis in the wall itself."

Traumatic Lesions; Endogenous Infection; Postoperative Infection: Focal infection complicating a traumatic lesion of the eye is occasionally encountered. I have had personal experience with several cases of this type. I recall

one, a patient who developed a destructive corneal ulcer following a slight trauma from a small corneal foreign body, which resisted the usual management of such lesions. It was only after I had a Wassermann done, with a positive finding, followed by instituting the appropriate specific treatment, that the ulcerative process was checked and went on to repair.

Fuchs states: "Endogenous infection consists in this, that pathogenic germs get into the blood and are carried with it into the vessels of the eye where they settle." Under the title, "Autotoxis," he observes: "Apparently more frequent are the cases in which various structural diseases of the eye, such as keratitis, chronic iritis, cyclitis, choroiditis and retinal hemorrhages, and also varous functional eye disorders (asthenopia, insufficiency of accommodation, and convergence) are caused by bad teeth or by disease of the tonsils or accessory cavities of the nose. Here, however, we have to do in most cases, not with the poisons produced by derangement of metabolism, but with bacterial toxins (endogenous infection)." He further states: "By endogenous infection of wounds we understand the lodgement of microbes, which are present in the circulating blood, in those parts of the body that have been injured by traumatism." "In animals," he continues, "an infection of this sort can be set up experimentally by injecting pyogenic germs into the blood channel and then injuring the eye; from some observations that have been carefully made, it is probable that the same process occurs at times in man."

In the literature, I find reports of very many of such cases, for example, postoperative infection following cataract extraction, the focus of infection of which were variously traced to the teeth, tonsil, nasal accessory sinuses, prostate, etc. As Knapp aptly expresses it: "Chronic septic foci frequently exert a baneful influence on eyes that have recently been operated upon. Various grades of iridocyclitis, which, in the writer's experience, developed in the second week after an uneventful cataract extraction, have been apparently due to tooth infection."

If opportunity permitted, many other ocular references appearing in the literature, as being dependent or influenced by focal infection, might be mentioned, such as choroiditis, cataract, vitreous opacities, keratitis, manifestations of syphilis and tuberculosis, the latter two being characterized by

Billings as "Classic metastatic infectious diseases." The literature pertaining to many of these entities is, however, so extensive, and the opinions as to their etiologic relationship to focal infection so varied, that justice could not be done them within the limits of my presentation. Sympathetic ophthalmia is of special interest. Key has reported a case of sympathetic ophthalmia cured by exenteration of the nasal accessory sinuses. Elschnig's and Alan C. Wood's researches on the anaphylactic theory of sympathetic ophthalmia are notable contributions to this puzzling disease, and are of special value to those interested in the study of focal infection.

The conclusions I would offer as a result of my investigation of the literature of this subject and my personal clinical experience are as follows:

1st. The "focal infection concept" as an etiologic factor in ocular pathology is an accepted scientific fact, established and supported by an abundance of clinical experience and experimental research.

2nd. Many sources of focal infection as factors in ocular lesions are recorded in the literature, but in my own investigations, the proponderance of case reports referred to the teeth, the tonsils, the nasal accessory sinuses, the deep urethra, and the prostate.

3rd. A survey of the subject points unmistakably to the wisdom of repeated and intensive cooperation—"team work"—on the part of the internist, the neurologist, the oral specialist, the laboratory worker, the roentgenologist, the rhinootolaryngologist and the ophthalmologist, in an investigation of obscure lesions of the eye. Without such cooperation, an intelligent scientific investigation cannot be conducted, and the failure to enlist it results in many instances, in needless sacrifice of visual function.

4th. The routine investigation of obscure ocular lesions by the ophthalmologist calls forth his every scientific resource, particularly in conducting the very necessary examination of the visual fields. And since the latter is a wholly subjective examination, the exercise on his part of the utmost patience in conducting this procedure is necessary to avoid incorrect interpretations or failure.

5th. The doctrine of focal infection, particularly in its application to ocular pathology, is a tremendously fascinating one. It promises much as an explanation of many obscure

ocular lesions. Under these circumstances, it is wise to preserve an open mind and balanced judgment. Overenthusiasm should not mislead one into the performance of ill advised operative measures, nor should overconservatism deter one from instituting procedures which a conscientious investigation dictates.

6th. Endogenous infection, traumatic ocular lesions, postoperative infection are subjects which present a very practical phase in the study of focal infection. A review of the literature impresses one as to the wisdom of subjecting patients afflicted with ocular trauma—operative or otherwise—who, under the usual management, are not exhibiting satisfactory progress, to a thoro investigation designed to establish the possible influence of a latent focal infection on the clinical progress of the case. In this connection, it should be emphasized that many focal infections may be "symptomless"; the patient is not aware of their existence, and no very evident pathology is disclosed. These types are especially perplexing and demand the utmost care in conducting the investigation.

It would seem also the part of prudence and good surgical judgment, in so far as is practicable and without inflicting ill advised hardship, to eliminate all sources of focal infection before subjecting a patient to a serious ophthalmic operation.

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DENTAL INFECTIONS AND THEIR SEQUELAE.

EARL C. RICE, D.D.S.

PHILADELPHIA, PENNSYLVANIA.

The dental phase of focal infection is replete with interesting findings, resulting from the cordial cooperation of the medical and dental men in their search for those truths that defy contradiction.

The dentist who has spent long years carrying out a painstaking technic in the treatment of pulpless teeth, is placed in a difficult position when confronted with the fact that his efforts have been but sparingly successful.

A very large percentage of the dental men do not recognize the grave danger of focal infection, and it will be found that these men do not cooperate with the bacteriologic laboratory, nor do they attach importance to the investigations of others who have gone farther afield than they.

While these men are sincere in the main, their opinions on the subject in hand fall far short of gratifying as dependable data in the great effort to definitely class dental foci of infection as etiologic factors of ill health.

Many dentists of today condemn all pulpless teeth to the forceps. This is a commendable prophylactic measure, since many, if not all, of these teeth are sources of infection, and only after extraction, can this be determined.

The roentgenogram is an indispensable adjunct in dental diagnosis, but its limitations cannot be too strongly emphasized. Roentgenographically a tooth may be negative, while bacteriologically it may be the seat of virulent infection.

We have reached a point in our search for the relationship between dental diseases and other bodily ills where "hit or miss" diagnosis cannot be countenanced.

In the opinions of most intellectual investigators, all pulpless teeth and fragments of retained roots must be looked

upon with suspicion, even tho they be symptom free and the patient in an apparent state of health.

The human body successfully carries many burdens of infection. If it were not for the vigilent activity of the protective armament within, we should all have died in infancy.

It has been said that specialization has a tendency to make the viewpoint narrow, but the truth of that statement is doubtful. The specialist has made his work a part of his soul's self, and thru his vast intimacy with it, he furnishes a beacon for the storm tossed general practitioner who has lost his way. It is thru specialization in the various fields of medicine that we each furnish a chapter in the story of professional effort, and it is safe to say that it not only broadens us but brings us closer together.

The diagnosis of dental foci of infection, is often attended with great uncertainty. Roentgenographic records point out apical rarefactions, fragments of retained roots, necrotic bone, osteitis, and so on. But many pathologic conditions exist to which pictures alone give no clue.

In the absence of symptoms, effort must be made to determine the condition of the dental pulp, for it is definitely known that vital pulps may be symptom free and yet pathologic, and not infrequently they respond normally to the various tests. Again, normal vital pulps give every evidence of being nonvital when tested. Hence the difficulty of an exhaustive diagnosis.

The greatest hinderance to tooth salvation is found in the fact that the success of the remedial effort can be definitely determined only in the pathologic laboratory, and that as a sequel of tooth extraction.

It would seem that the best policy is to assume that the teeth are in a state of ill health until they can be proven otherwise.

The statements that have been set down here are based upon records of many hundreds of cases where pathology of the various organs of the body have completely cleared up after the extraction of infected teeth, many of which were symptom free, and not a few when tests revealed no disorder.

It will be interesting to present the following list of known cases:

Myocarditis.	Ptosis.
Endocarditis.	Loss of vision.
Cardiac arythmia.	Sinusitis.
Cholecystitis.	Arthritis.
Cystitis.	Myositis.
Nephritis.	Bell's palsy.
Corneal ulcer.	Dermatitis.
Iritis.	Neuritis.
Glaucoma.	

Altho the removal of teeth as a remedial measure has met many failures, yet it is a prophylactic step that must be given credit for saving many lives, and bringing comfort to legions of afflicted humans, and no one can be entirely safe with one infected tooth in situ.

TRANSACTIONS

OF THE

THIRTY-SECOND ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OPHTHALMOLOGIC SESSIONS

VISUALIZATION OF THE LACRIMAL PASSAGES BY THE USE OF LIPIODOL.

MARY KNIGHT ASBURY, M.D.

CINCINNATI, OHIO.

The use of opaque substances for visualization of the lacrimal passages in roentgengraphy was first reported by Ewing of St. Louis, in 1909. He injected abscessed lacrimal sacs with a mixture of finely sifted bismuth in albolene, and then took two temporal views of each case; one with a lacrimal probe in place, and one without, to show the relation of the duct to the dilated sac.

Two years later, Aubaret, without knowledge of Ewing's work, reported similar investigation of a number of lacrimal lesions. He used mixtures of lead or bismuth in paraffin oil, and took radiograms in several positions of the head. Aubaret's work covered a much wider field than Ewing's, and he demonstrated cases of stenosis, fistula and false passage, as well as ectasia of the sac.

Both communications must have remained rather obscure, for in 1914, von Szily reported as original, work along a similar line. He tried a number of radioopaque substances, but finally chose a mixture of thorium in liquid paraffin as the most satisfactory. He wrote several articles on the roentgenographic investigation of the lacrimal passages, and really brought the matter to the attention of ophthalmologists. All of the later articles, of which there is a number in the foreign literature, refer to the original work of von Szily.

In 1921, Sicard and Forestier began using lipiodol as a constrast substance in roentgenologic diagnosis. Lipiodol is an iodized vegetable oil (poppy oil), containing 40% iodin by weight. It is not a solution of iodin in oil, but a chemical combination, which gives no reaction for iodin by the ordinary methods. Iodized oil had been used therapeutically for some time, and several observers had noted the dense shadow cast by the substance in roentgenograms after it had been injected subcutaneously or intramuscularly. It was also

known that altho lipiodol was absorbed slowly in the tissues, it was nonirritating, and did not give rise to tumor formation. Sicard and Forestier were the first to make use of this knowledge. They used lipiodol in exploring the epidural and subarachnoid spaces of the spinal canal, in the bronchi, and in old abscess cavities and fistulous tracts. In 1923, Bollock, one of their colleagues, used lipiodol to visualize the lacrimal passages in roentgenograms.

The number and variety of contrast substances used for visualizing the lacrimal passages in the past, is an indication that none was entirely satsfactory. The particulate substances, such as the bismuth and barium mixtures, were hard to inject, harder to eliminate after the picture had been taken, and cast poor shadows in the minute quantities required by the normal lacrimal passages. Some mixtures had to be made up fresh for each injection, and all had to be sterilized. Lipiodol overcame many of these objections. It is unusually opaque to X-rays. It is a homogenous substance, and altho it is a thick oil of high specific gravity (1.350), it is sufficiently

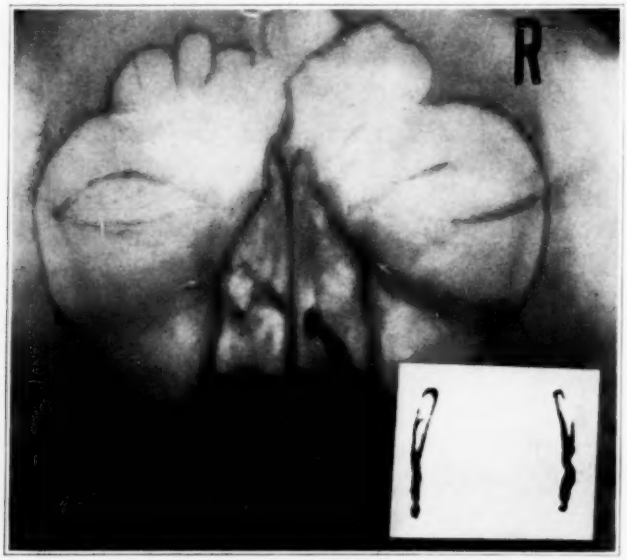

Fig. 1. Four minutes elapsed between the injection and the X-ray; 0.5 c.c. used. The oil has flowed on thru the sacs, but enough adheres to the walls to outline them. Palpebral fissures outlined by the oil. Constriction below the sac more marked on right side.

fluid at body temperature to flow into and out of the lacrimal duct, and it can be warmed to increase its fluidity without destroying the compound. Altho lipiodol is fairly stable, it is not advisable to sterilize it at high temperatures or to submit it to sunlight for long periods. Sterilization is not necessary, for Neuswanger found that iodized oil exposed to the air for several months showed no evidence of decomposition, and no colonies of bacteria or fungi were observed. However, its bactericidal power is a debated question. Forestier claims lipiodol has this action, but gives no proof. Experiments conducted by Neuswanger and by Archibald seem to refute this claim. Globules of oil in close proximity to colonies of various bacteria on agar plates exerted no inhibitory action on the growth. On the other hand, pathologic conditions sometimes seem to be improved following the injection of lipiodol, for instance, the foul sputum of bronchiectasis often loses its odor. Lipiodol is nonirritating to mucous surfaces, an advantage shared by few of the contrast substances previously used. None of my patients complained of the injection, and none had any objective symptoms of conjunctival irritation following injection. I have also used lipiodin (Ciba), which

Fig. 2. Lateral view of same case showing injected ducts side by side. Constriction plainly seen. Excess oil trickling into pharynx.

substitutes sesame oil for poppy oil. It gives as good shadows as the French lipiodol, and seems slightly easier to inject. My samples of lipiodin turned cloudy shortly after opening, so that I was afraid to use it on a second case, but cultures showed no bacterial growth. Clouding probably was due to some chemical disturbance in the oil.

TECHNIC: Both lacrimal passages are routinely injected for comparison. It is advisable first to instill a drop of cocain solution or other local anesthetic at the inner corner of each eye. For the injection, an ordinary lacrimal needle and Luer syringe are used. The syringe is warmed so that it will not cool the oil when filled. Considerable pressure is necessary to inject the warm oil thru the small caliber lacrimal needle, and it is almost impossible to force cold oil thru such a needle. The oil is best warmed by immersing the container in hot water for a few minutes. It must be clear yellow. If it is cloudy or brownish, it contains free iodin, and must not be used. Injection is made thru the lower punctum, unless obstructed, when the upper can be used. 0.5 c.c. will more than fill an average duct. 0.2 c.c. will clearly outline a normal one. An excess of oil does no harm, and when it flows back into

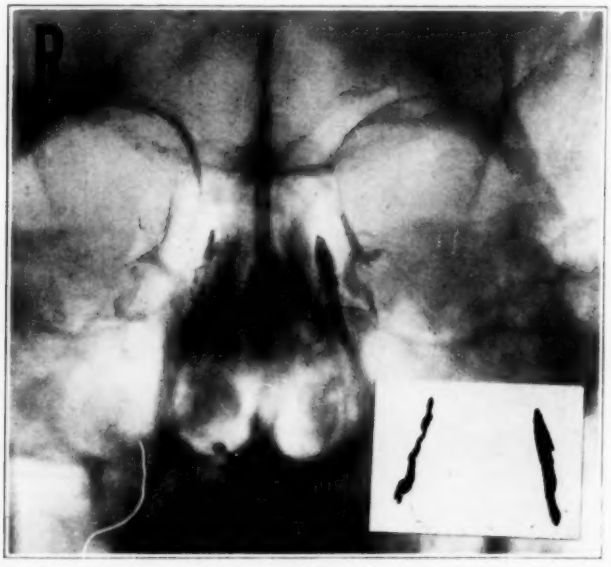

Fig. 3. Lacrimal passages irrigated with boric solution before injection of lipiodol. Left side injected first (0.3 c.c. used).

the conjunctival sac, is an aid, for it then outlines the palpebral fissure and sometimes the canaliculi and their junction with the sac. (Fig. 8.) As a rule the excess flows into the pharynx and is swallowed. (Fig. 2.) Altho the iodin is rapidly freed and absorbed in the gastrointestinal tract, there is no danger of iodism from the minute quantities used.

Since lipiodol runs thru the lacrimal canal very quickly, X-ray pictures must be taken immediately after injection. It was found best to have the patient lie down on the table where the roentgenograms are to be made, before injecting the oil. If both lacrimal passages are patent, one should be injected and the film exposed before the other is filled. If one passage is obstructed, both can be injected before taking the picture, but the obstructed side should be injected first.

Most foreign authors recommend irrigation of the lacrimal passages before injection of the contrast substance. Cases complaining of epiphora are routinely irrigated to determine whether there is obstruction. Sometimes irrigation preceded injection of lipiodol. (Fig. 3.) In other cases it had been done on another day (Fig. 4). Comparing plates, preliminary washing did not seem to increase the clarity of the shadows.

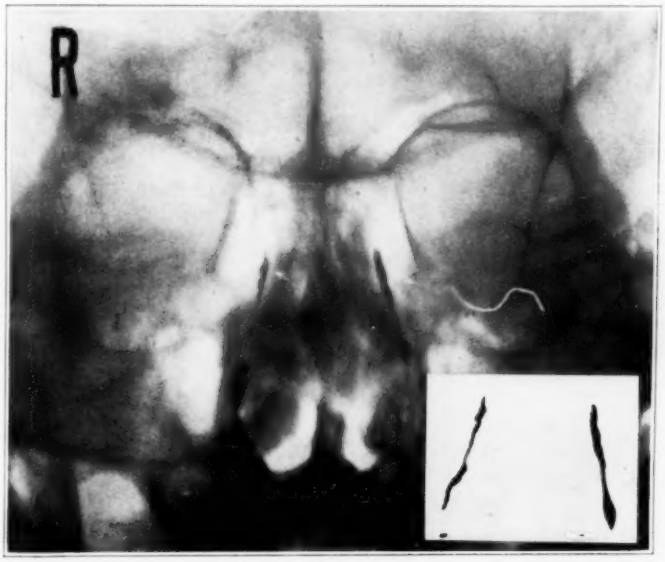

Fig. 4. Same case ten days later. No preliminary irrigation; 0.5 c.c. injected. Note decrease in caliber of passages. Patient complained of epiphora of right eye.

It is important to thoroly express a dilated sac before injecting, because accumulated pus or tears prevent the contrast substance filling the entire cavity.

Roentgenograms are taken in both frontal and lateral positions. The frontal view is best taken with the forehead and nose touching the plate, for then the shadows of the lacrimal passages are thrown over the thinner ethmoid region. (Fig. 5 and 6.) Lateral views are made at a slight angle, so that the two shadows will not be superimposed. This roentgenologic examination presupposes close association between the roentgenologist and ophthalmologist or access to an X-ray laboratory, for it is almost necessary that the ophthalmologist inject the lipiodol himself, but the information derived from this special examination makes it worth the extra effort. The X-ray picture itself, without the contrast substance, is of value in determining the state of health of adjoining structures, which is of etiologic significance. The injection of the oil outlines the lacrimal passages so that their caliber can be noted, the level of obstruction determined, and dilatation of the sac, fistulae and diverticula seen. This information aids in choosing the proper kind of treatment as well as elucidating the cause of epiphora.

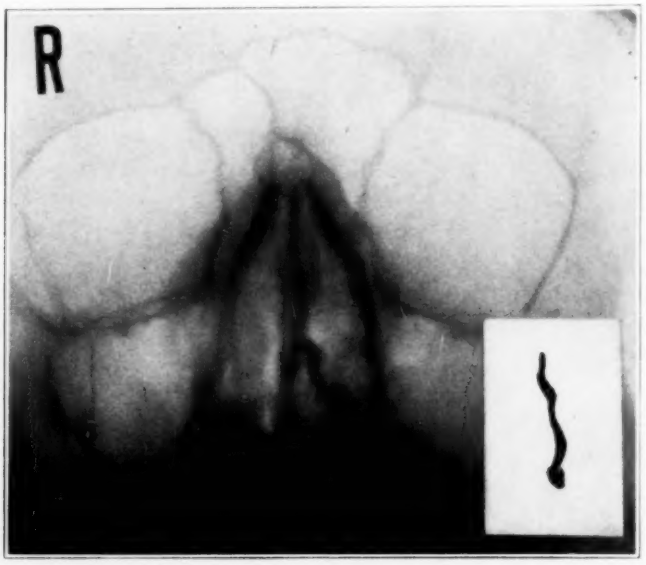

Fig. 5. The left passage only was injected, 0.3 c.c., the X-ray picture taken immediately, the nose and chin resting on the plate.

One patient had been relieved of severe epiphora by probing, but occasionally the symptoms recurred in a mild form. The X-ray showed a narrowing of the passage at the level of the junction of the sac and the nasal duct. (Fig. 1 and 2.) Irrigation and an astringent collyrium always relieve the epiphora. It is easily conceivable that a slight swelling of the mucosa at this point would cause temporary obstruction.

An interesting observation was made in one patient on whom roentgenologic examination was repeated in ten days. In the first roentgenograms, the left duct was dilated, the right very narrow. (Fig. 3.) Ten days later, the left duct was about normal width, the right still narrow. (Fig. 4.) The patient complained of transient epiphora, affecting first one, then the other eye. At the time of examination the right eye, with the narrow passage, was weeping.

A man of middle age had had mild epiphora for a number of years. In the past the lacrimal ducts had been probed on several occasions, and the right canaliculus had been slit.

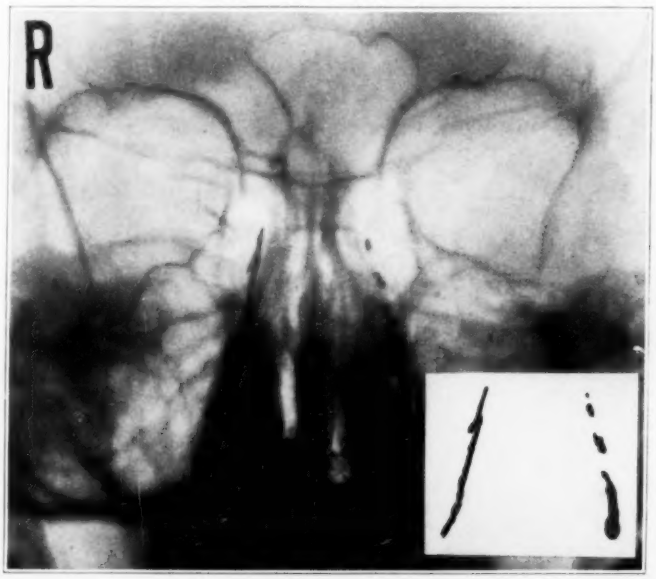

Fig. 6. (Same case as Fig. 5.) As soon as X-ray, shown on Fig. 5, was taken, the right passage was injected and another exposure made. Upper part of left passage already empty. Small diverticulum of right sac. Picture taken with forehead and nose touching plate.

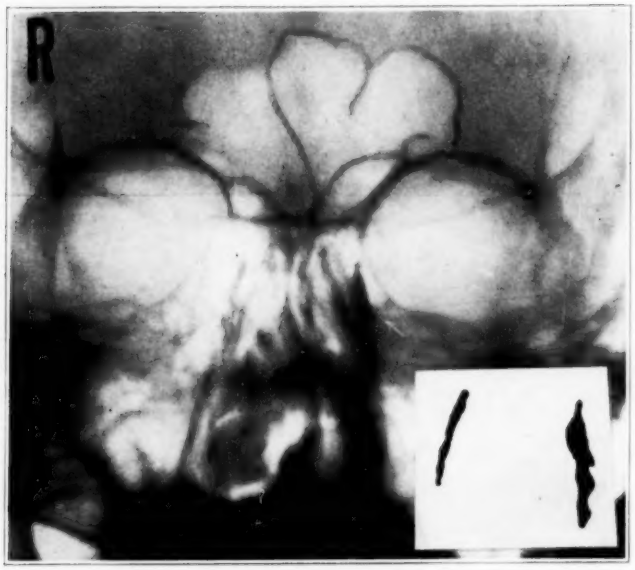

Fig. 7. Dilatation of the left lacrimal passage. Patient had no epiphora or eye symptoms of any kind. Lipiodin (Ciba) 0.5 c.c. used.

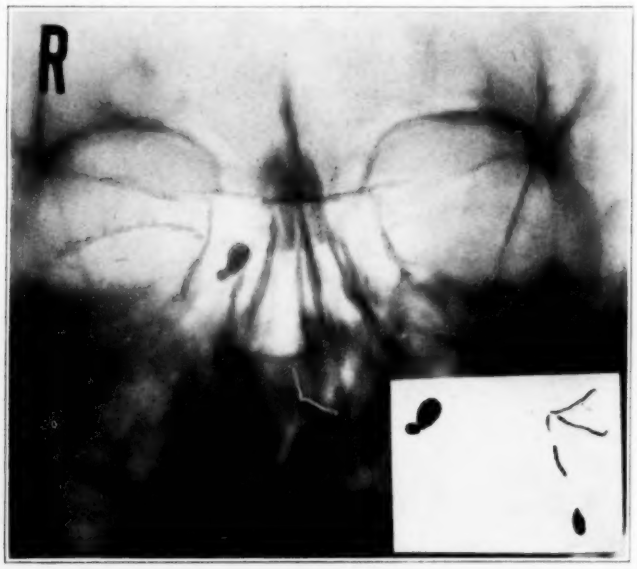

Fig. 8. Chronic purulent dacryocystitis, right side. Abscess sac filled and palpebral fissure outlined with lipiodol. Left canaliculi filled with oil. Oil drop in left side of nose, none in right side.

There is a small diverticulum of the right sac, possibly produced by false probing. (Fig. 6.) Another patient who had no epiphora or symptoms referable to the lacrimal apparatus was injected to determine a point of technic. The roentgenograms showed marked dilatation of the left lacrimal duct. (Fig. 7.) No explanation of this anatomic anomaly is offered, but it illustrates the difficulties encountered in interpreting findings.

The choice of an operation for chronic purulent dacryocystitis can be made more rationally when the size of the sac and the location of the obstruction as shown by lipiodol injection are known. If intranasal drainage is contemplated, it is necessary to have a roomy sac. Roentgenograms after injection of iodized oil give the most accurate information on this point.

Figure 8 shows an abscessed sac in which the obstruction is high. The duct was probed several times without benefit, and then the sac extirpated. This patient had a diseased nose, which is always a contraindication for intranasal drainage.

Figures 9 and 10 show the result of a Mosher-Toti type of operation in a patient who had a nose full of pus. The

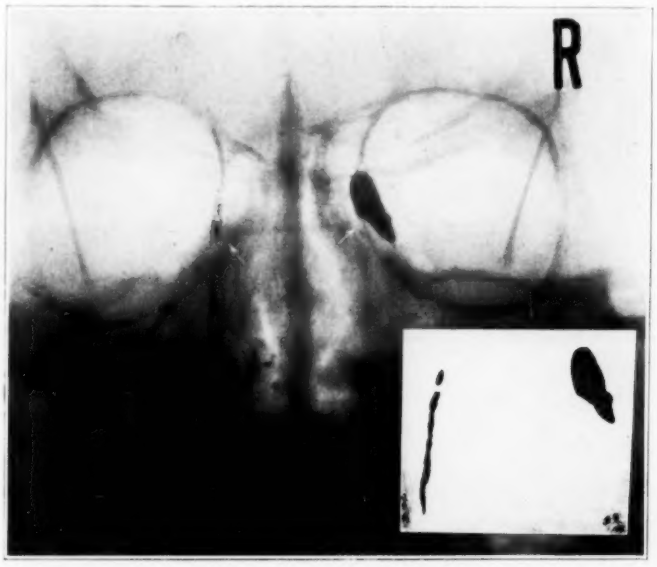

Fig. 9. Large abscess cavity right side. Left lacrimal passage patent.

opening into the nose is closed. There is a large abscess cavity and a fistulous opening in the scar on the cheek, thru which thin purulent secretion constantly oozes.

Conclusions.

1. The injection of a contrast substance is a valuable aid in diagnosis of, and in the choice of suitable treatment for lesions of the lacrimal passages.

2. Iodized oil is the best contrast substance which has been used for visualizing the lacrimal tract. It is very opaque to X-rays, nonirritating to mucous membrane, and the technic of injection is simple.

I wish to express my gratitude to Dr. Samuel Brown, roentgenologist, whose cooperation made this work possible.

Fig. 10. Lateral view of same case showing fistula from abscess cavity onto cheek.

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DISCUSSION.

Dr. Lawrence Post, St. Louis, Mo.: Perhaps the most unwelcome cases that come to the ophthalmologist are those of chronic dacryocystitis, because the handling of them is so unsatisfactory. The condition of slight tearing with occasional matter in the eye is not sufficiently alarming, and too insidious in origin, to cause patients so troubled to seek advice early, therefore the process is well established when they are first seen. They tend to regard the trouble as a nuisance rather than anything serious, and prefer tolerating it indefinitely to submitting to any painful or radical procedure.

It used to be my practice to attempt, first, the syringing of fluid thru the canaliculi into the nose, and then the passage of probes of gradually increasing size. Temporary improvement was the rule, but lasting improvement the exception. Underlying nasal pathology was always sought, and when found, was treated. I am strongly of the opinion that nasal pathology is the basis of more than ninety per cent of the cases, and that there is no hope of a cure unless the nasal pathology is eliminated. In many in whom probing has proved unsatisfactory, I have removed the sac.

I have always felt that I did not know as much about the lacrimal passage that I was treating as I should like to know, and was continually on the lookout for some method of increasing the accuracy of diagnosis.

In 1909, my associate, Dr. A. E. Ewing, had roentgenograms made of the lacrimal passages injected with bismuth paste, but tho the pictures thus obtained were good, the disadvantage of the paste, especially in that it could not be readily removed, was obvious, so that the method was not continued.

A knowledge of the exact size and localization of the sac was desirable, but a suitable substance for injection was not found for several years.

Lipiodol injections of the lacrimal sac were reported by Bollack in May, 1924. The substance is an iodized vegetable oil containing forty per cent of iodin. It is clear, straw colored and quite thick. It shows very distinctly in Roentgen ray pictures, and when warmed, is readily injected and quickly eliminated from a normal lacrimal apparatus. It is,

therefore, ideal for visualization of the sac and determination of its function.

I first used lipiodol in June, 1925, having procured the drug from the Lafay Company, which supplied the original workers, and for the past two years have used it in most of my cases of dacryocystitis.

The oil is dispensed in small ampules. After emptying the sac by pressure, the ampule is warmed slightly and about .5 c.c. of the fluid injected by means of a syringe made by fusing a platinum needle, with a bore of about .6 mm., into an ordinary dropper. The upper punctum is obstructed and the lipiodol injected. Excess of fluid is wiped from the face and conjunctival sac. The patient then puts his face squarely against the Roentgen ray plate, the forehead and top of the nose resting on the plate, and the exposure made.

A lateral view may also be taken. A second picture a few minutes later will show the rate of excretion. The sac and nasolacrimal duct are thus clearly defined, and the knowledge so gained may be used as an indication for future therapy.

The employment of this method for only two years, in a limited number of cases, does not permit me to speak with great assurance, but my experience has been, that in those cases where a much enlarged sac was found, probing with small and medium sized probes—I have not used larger than Bowman No. 8—has not been effectual. Recently, therefore, I have not undertaken such treatment in this type of case but have advocated extirpation of the sac. The Roentgen ray picture has here eliminated the long, painful and useless treatment with probes. On the other hand, where the sac is shown to be still small, and the elimination time not greatly above normal, probing is apt to prove successful.

I cannot speak for the value of lipiodol in determining the results of nasolacrimal operations, as I have never performed these operations. Tho extirpation of the sac is not what might be called a physiologic procedure, but it certainly is a reasonably satisfactory one, and rarely have I been forced to excise the accessory lacrimal gland for postoperative tearing, but when it has been necessary, it has proved efficacious.

Where external fistulae are present, my practice has been to excise the fistulous tract and cauterize the sac with trichloracetic acid. Except for a somewhat larger scar, the results from this treatment have been as good as from complete extirpation of the sac. Either operation can be performed under local or general anesthesia.

If a study over several years of a sufficient number of cases will show that the size, position and date of drainage of the lacrimal apparatus are true indicators of the best form of treatment, lipiodol, which is excellent for making these determinations, will have established its place in ophthalmology.

Dr. L. V. Stegman, Battle Creek, Mich.: This is a roentgenogram of a case of dacryocystitis in a man 70 years of age, with very friable tissues and large bored canaliculi. A pledget soaked in pure strength lipiodol was placed in the inferior meatus and removed, but enough remained to outline the vault of the meatus. After cocainizing the canaliculus and sac, a No. 6 Bowman probe was passed thru the upper canaliculus, sac and nasal duct with difficulty, and another probe was passed

into the inferior meatus, where they met. After the roentgenogram was taken and the probes were removed, 2 c.c. of full strength lipiodol was slowly injected thru these passages in order to fill them more completely. The second roentgenogram was then made. There was no further irrigation. The patient returned in one week, when the lacrimal passages were irrigated with boric solution, with the removal of globules of oil. For the first time, the probes were passed easily to the floor of the nose. It seemed to us that the oil carried out the mucus better than a watery solution, and that it had held apart the granulations, so that for the first time after probing they had not adhered.

The patient complained of an irritation during this week, which reaction we believed was due to the iodin that was liberated by light.

I have used the iodized oil in only two cases. One was an infant nine months old, with obstruction of the nasal duct from birth, who had been subjected to treatment since two weeks old at various clinics in China. There was a fistula in the cheek, from which much mucopus discharged.

The other case was the old man of 70. In each case the passage of

Fig. 1. Left lacrimal sac injected with lipiodol. Normal. (Daily.)

the probes was made easily, and the canals remained patent after the irrigation with lipiodol.

The last slide shows that the line drawn along the globules of oil superimposes upon the probes when in position.

DR. ALLEN GREENWOOD, Boston, Mass.: I want to congratulate Doctor Asbury on her work, and to call attention to the fact that here you have something that will aid you in acquiring that delicacy of touch that is necessary in the use of probes.

Doctor Stegman said something about the case of an infant. I want to make it emphatic, as I have every time I have spoken on this subject, that infants should not be probed. There is no operation I know of that requires more accurate knowledge of what you are going to do, and especially knowledge of direction of the lacrimal passages, and this you can get from the X-ray method of examining the patient which has been brought out by Doctor Asbury.

DR. RAY K. DAILY, Houston, Tex.: We have been using roentgenograms of the lacrimal passages injected with radioopaque substances, as a diagnostic procedure, for some years. We use bismuth paste, which

Fig. 2. Right sac injected with lipiodol, was operated intranasally eight months ago by the Hall-West Method. Lipiodol enters the nose above the inferior turbinate.

is easily made from bismuth subnitrate powder and liquid albolene; after the X-ray is taken, the sac is irrigated with boric acid. We have never seen any irritation or other untoward symptoms from its use. We used lipiodol after it came out; it has no advantage over bismuth paste, and the rapidity with which it flows thru into the nose, makes it necessary to make the injection in the X-ray laboratory, which is inconvenient to say the least. So we have returned to the use of bismuth. Such an examination is of value in making the diagnosis and determining the course of treatment in dacryocystitis. Given a case of purulent dacryocystitis, one is justified in advising immediate operation. We prefer an intranasal operation upon the lacrimal sac as the first procedure; where it fails, one can always do a complete extirpation, but an intranasal operation leaves the lacrimal passages functioning, and is successful in so many cases, that it is hardly fair to the patient to extirpate the sac without first making an attempt to restore physiologic function. In

Fig. 3. Bismuth injection. Right side was operated intranasally one year ago; left side shows stenosis and dilatation above it.

cases of epiphora, such an examination shows whether we are dealing with a functional epiphora or pathology in the lacrimal passages; if the X-ray shows a stenosis, an intranasal opening into the sac, in front of the middle turbinate, will nearly always give relief. If the X-ray shows the passages normal, all treatment is futile and the patient will be spared a period of useless and painful probing.

The photographs of X-ray plates will show the difference in the lipiodol and bismuth plates.

1. Left lacrimal sac injected with lipiodol. Normal.

2. Right sac injected with lipiodol; the Halle-West intranasal operation was done eight months ago. The lipiodol enters the nose above the inferior turbinate.

3. Bismuth injection: Right side was operated intranasally one year ago; left side shows stenosis and dilatation above it.

4. Case of functional epiphora in a young woman who has complained of epiphora for years, and has been treated with lavage and dilatation. The plate shows normal lacrimal passages, which accounts for the futility of the treatment she had. Frontal view. Bismuth injection

5. The same case; lateral view.

Fig. 4. Case of functional epiphora. Both sacs injected with bismuth paste. Frontal view.

Fig. 5. Same case, lateral view. Functional epiphora. Bismuth injection.

DACRYOCYSTITIS: TREATMENT OF CHRONIC.

F. W. Dean, B.S., M.D., F.A.C.S.

COUNCIL BLUFFS, IOWA.

The treatment of few diseases in ophthalmology has given more disappointing and unsatisfactory results than cases of chronic dacryocystitis. The outcome is indeed so unsatisfactory, that of late rhinologists are opening and draining the lacrimal sac by the intranasal method. Some ophthalmologists are advocating that these cases be turned over to the rhinologists for treatment. I am opposed to such transfer. The eye is in more danger of becoming infected if an opening is made directly into the sac from the nasal cavity. Also, the intranasal operation is not by any means always successful, and perhaps a stronger reason is the unpleasant feeling that one has failed and given up.

The method of treating chronic dacryocystitis has not changed materially since Bowman introduced the use of his probes. The only change of any consequence is the increase in the size of probes employed by some ophthalmologists. H. D. Noyes of New York and E. Williams of Cincinnati were the first advocates of large probes. Schweigger, in Handbook of Ophthalmology, published in 1878, says that Bowman's larger probes are too large and must not be used, as they will injure the canaliculi. If probings fail, he advises obliteration of the sac with cautery or caustics.

Edouard Meyer, in Practical Treatise on Diseases of the Eye, published in 1887, outlines about the same treatment, tho he sometimes used larger probes.

Theobald, in 1877, made a study of the size of the bony lacrimal ducts in a number of skulls, and designed a set of probes up to size 16, which is 4 mm. in diameter. The late Dr. Ziegler was an enthusiastic advocate of the large probes. Both Theobald and Ziegler, however, slit and passed these large probes thru the lower canaliculi. Less harm is done if such operations are confined to the upper canaliculi.

Paul Roemer in Textbook of Ophthalmology, page 426,

says that he uses small probes and in addition makes use of small styles left in the duct between probings. He estimates that 5 or 10 per cent get permanent relief by this method. On page 428, he makes this flat statement, "The treatment of chronic catarrhal dacryocystitis is extirpation of the sac". Extirpation of the sac gives relief from septic troubles, but it does not do away with the annoying epiphora. I am not attempting to review the literature on chronic dacryocystitis, but have referred to the above authors to point out that the methods of treament have not changed materially during the last fifty or sixty years.

For probing the lacrimal ducts, some ophthalmologists still insist that small probes be employed; others contend that only the large probes will secure the desired results. The controversy would soon be settled if only either group were successful.

To treat chronic dacryocystitis successfully, there are three results that must be secured. First, free drainage of the sac; second, a sac rid of infection and granulations; third, puncta and canaliculi left in a normal condition.

The users of the small probes do not as a rule injure the puncta and canaliculi, nor do they establish free drainage of the sac. The users of the large probes establish free drainage, but more or less mutilate the puncta and canaliculi. It is very difficult to rid the sac of infection and granulations by treatments if attempted thru the canaliculi. The failure to put the lacrimal sac in a healthy condition is probably the cause of a large per cent of failures in the treatment of chronic dacryocystitis. To accomplish the three necessary tasks—the securing of free drainage, a healthy sac, and normal canaliculi and puncta, I am using the following procedure.

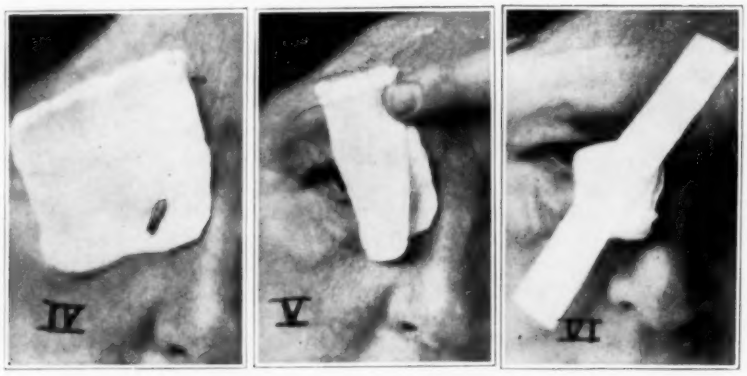
If fluid can be forced thru the canaliculi by pressure over the sac, they are patulous and need no attention. If they seem to be closed, a No. 4 Bowman probe is passed thru the upper and lower entrance into the sac, to make sure there are no strictures. A No. 4 probe will pass thru the puncta and canaliculi without injuring them, if proper care is taken.

After anesthetizing the tissues, an opening is made into the sac thru the skin, beginning at the lower border of the ligamentum orbicularis and extending downward about 7 mm. To anesthetize the sac preparatory to curetting or probing, a small gauze wick dampened with a 10 per cent solution of

cocain and a drop of epinephrin added is packed into the sac and as far down the nasal duct as possible. In five or ten minutes the anesthesia is complete. The pack is removed, and any granulations present are carefully removed by curettements. It is unusual to find granulations. The sac is wiped out with a 2 per cent solution of silver nitrate or any remedy suggested by the condition of the lining membrane. This surely is better surgery than to attempt treatment thru the canaliculi.

To secure free drainage of the sac, the lacrimal duct is dilated with large probes. Four probes are used; Ziegler's Nos. 10, 12, 14, and 16, which are 2½, 3, 3½, and 4 mm. in diameter. I have separated the Ziegler probes by cutting the handles in two in the middle. In this way, they are made less clumsy, and also the probable contamination of one end of the probe while introducing the other end is eliminated.

First, a No. 10 is passed thru the skin incision into the sac and down the duct. This is followed successively by the larger sizes, until the largest probe is passed that the duct will admit. Usually a No. 16 can be passed easily, tho some ducts will not admit a probe larger than a No. 14. There is no danger of making a false passage with these large probes, especially as the upper end of the duct is immediately under the opening made into the sac. After dilating the lacrimal duct with the largest probe possible, a silver wire about 2½ mm. in diameter is passed into the duct and a dressing applied. The upper one cm. of the wire is bent, so that it forms an angle of about 45 degrees with the shaft which enters the duct. This upper portion of the wire lies close to the skin below the incision and prevents the wire from becoming dislodged. Every second day, the sac and duct are anesthetized, the sac is treated thru the incised opening, the larger probes are passed, and the style and dressing replaced. As soon as the sac is in a healthy condition, the incised opening is allowed to close. Four or five treatments is the average number required to put the sac in a condition to allow the closing of the incision into the sac. With a sac in a healthy condition and having free drainage thru the lacrimal duct, there is a positive assurance that there will be no resultant fistula. In two cases, I used a piece of rubber tubing instead of a silver style. There was so much pain in each case, that it was necessary to remove it and substitute a silver wire, which has not caused severe pain in any case.



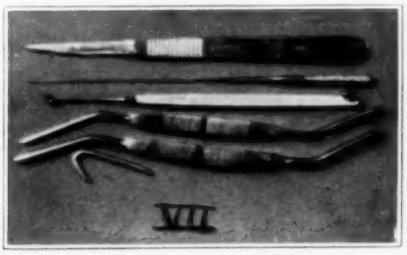


Fig. 1. Schematic drawing of canaliculi, sac and duct (Gray), showing location of opening into sac and direction of attack.
Fig. 2. Applicator for wiping out sac.
Fig. 3. No. 16 (4 mm.) probe in nasal duct.
Fig. 4, 5 and 6. Method of dressing with 2½ mm. wire style in nasal duct.
Fig. 7. Instruments used in operation, showing Ziegler probes cut in two.

Of late, I have had made a silver style 2½ mm. in diameter, the shaft of which is hollow. The inside diameter of the tube is 1½ mm. Theoretically this tube should keep the sac drained and bring about a cure sooner than the solid style. It does drain the sac, but I have not used the hollow style in a sufficient number of cases to determine the extent of its superiority.

DISCUSSION.

DR. C. S. O'BRIEN, Iowa City, Iowa: Restoration of the natural lacrimal passages is undoubtedly the ideal treatment in chronic dacryocystitis.

Ware, in the latter part of the eighteenth century, advocated opening into the sac thru an incision below the internal palpebral ligament. This was followed by the insertion of a nail headed style thru the nasolacrimal duct, the flared style head being allowed to remain outside. The style was allowed to remain in the duct a few days, and was then removed and the skin incision allowed to heal.

Some authors state that J. L. Petit was the first to suggest temporary styles. Petit made an incision into the lacrimal sac, passed a grooved director thru the nasolacrimal duct, and introduced a bougie. This bougie was changed daily over a short period of time, then removed and the skin incision allowed to heal.

Dupeytren (1777-1835) revived an operation described by Wathen in 1781. In this operation, following incision into the sac, a gold or silver canula was passed into the nasolacrimal duct and allowed to remain. The skin incision was closed, and it was hoped that the style would remain permanently, but sooner or later most of them came out and the stricture recurred.

Seemingly the above mentioned methods fell into disrepute, due to the fact that the results were not permanent, it being stated that the strictures recurred following loss of the canula, even tho it had been in as long as fifteen to twenty years. Naturally, one would expect a temporary canula, such as those advised by Ware, Petit and Doctor Dean, to produce even fewer lasting results than a more or less permanent one. Perhaps Doctor Dean has uncovered the secret in his more thoro dilatation of the duct with large Ziegler probes, and the treatment of the sac with silver nitrate.

I have been able to perform the operation as advocated by Doctor Dean in only two instances. These were done shortly after I had talked with him, and since reading his paper, I realize that my technic was not what it should have been. The results were not satisfactory, probably due to the fact that the operation was not properly carried out. I shall try again and adhere rigidly to his technic, since I feel that the procedure is ideal if it will produce results. It is simple, rapid, and if the results are permanent, it seems to me that it could not be improved upon.

At present, our method in the University of Iowa is the usual extirpation of the sac, accompanied very often by resection of the accessory lacrimal gland. This operation allows our patients to leave the hospital after

about four days, and the results, while not ideal, are quite good, since there is no epiphora and the focus of infection has been removed.

Dr. Delos W. Hogue, Springfield, Ohio: The treatment of a closed lacrimal duct has been for the most part unsatisfactory. The majority of the methods that have been suggested in the last twenty years have had some merit, altho none of them have given perfect results in every case. I have never approved of the removal of either the gland or the sac, as it has always seemed to me to be a confession of our inability to cope with this troublesome condition in a proper surgical manner.

I have had the best results from the method described by Fraser, in the Journal of Laryngology, Rhinology and Otology, 1914, and in most cases have secured excellent results. I thoroly approve of Dr. Dean's suggestion that we eliminate as much as possible any injury to the puncta and canaliculi. It is very essential to the success of any operation for draining the lacrimal sac into the nose, that these be left in normal condition.

Another point which is an excellent suggestion, is the removing of any diseased tissue in the sac, and this could be done by this method.

There are some cases in which it is almost impossible to keep the lacrimal ducts open by means of probing, due to their tortuous course, and in such cases I can see no objection to doing the Fraser operation thru the opening which Dr. Dean makes into the sac.

I have found, in a few cases, that the nasal opening will close after doing the intranasal operation, and I have recently devised an instrument which has been of great help in these cases. I will show this tomorrow when the new instruments are demonstrated.

Dr. James M. Patton, Omaha, Neb.: This Academy is indebted to Dr. Dean for a number of very helpful suggestions, and his technic relating to the care of this type of tear sac trouble is not the least of his contributions. If we could find some sort of operation that would take care of all cases of dacryocystitis, we would be fortunate, but so far this has not been found. However, this operation suggested by Dr. Dean does care for a number of cases which are exceedingly difficult to treat in any other way, unless we mutilate the canaliculi and puncta, and while, as Dr. Dean has suggested, mutilated puncta and canaliculi with drainage are undoubtedly better than intact structures without drainage, still if we can operate without mutilating these structures, so much the better.

In cases where it is desirable to secure prompt drainage, I have cut down on the sac, put a wire thru into the nose, and after this have tried dilation, which Dr. Dean has emphasized. While we have not used it in very many cases, there is no question that it is of real value.

One of the gentlemen, in discussion, spoke about destroying the sac by dissection, and stated that it required three or four days of hospitalization. I can not refrain from reminding you that if you do not like the dissection operation, and if you are in a hurry, or if for any other reason you do not wish to do the extirpation operation, you will find that the trichloracetic acid method as advocated by Dr. Harold Gifford does meet the requirements, and hospitalization of the patient is not necessary. The procedure is very simple and the results satisfactory.

Dr. F. W. Dean, Council Bluffs, Ia. (closing): My strongest reason for not wholly enjoying my operation is that I like to dissect out the sac.

I do not like to use trichloracetic acid or cause any inflammation when I can dissect out the sac and have so much fun doing it.

I do not like the intranasal method, not only because of the danger of infecting the eye, but I would hesitate quite a little before I would allow anyone to make an opening into my nose—open the anterior ethmoids and pour pus in from the lacrimal sac. I think that is a bad procedure.

This method of treating by dilation of the duct and cleansing the sac, is one that can be carried on in the office and have the patient return every second day. It is not necessary to confine the patient in the hospital at all.

OCULAR LYMPHATICS.

JOHN F. FULTON, M.D.

ST. PAUL, MINNESOTA.

All that one can accomplish in the brief time allotted to this paper, is to draw your attention to the fact that the anatomy and purpose of ocular lymphatics are in doubt, and to express the hope that the research worker in the very near future will give them the special attention that they richly deserve.

No subject in ophthalmology is more confusing and unsatisfactory than the study of the literature which deals with the origin and nature of the intraocular fluids, and the vessels and spaces which contain them. It is characterized by nonlogical theories, acrimonious debate, and clumsy experiments devoid of useful results; all of which demonstrate lack of accurate knowledge of the vessels that distribute lymph to the various parts of the eyeball. I am quite in sympathy with a well known English author, that it is difficult to emerge from the perusal of this with an unsoured disposition.

W. Stewart Duke-Elder, in a recent very elaborate and highly meritorious paper on the nature of intraocular fluids, states that lymph is the fluid contained in an anatomically closed system, which seems to be altogether unrepresented in the eye. At the same time, this author, in a recent paper on ocular circulation, defines capillaries as simple endothelial tubes thru which fluid interchange can take place. There is probably no sharp dividing line, either anatomically or physiologically, in structure or in function between them and the arterioles on the one hand, and the venules on the other.

This, according to F. E. Lee, K. W. Dewey and many others, is a description of lymphatic vessels. They claim that lymph is a product of endothelic activity. This I accept as having been established, and will publish with this paper a bibliography which seems to me to demonstrate this satisfactorily.

After having made a careful survey of the literature, I am

quite prepared to agree with Sabin that there are definite lymphatic capillaries and tissue spaces, and that it clears the situation greatly by remembering that plasma is fluid within the blood vessels, lymph is fluid within the lymphatics, and tissue fluid is fluid in the tissue spaces. The lymphatics having a specific function as absorbents, play a most important role, when normally functioning, in maintaining intraocular pressure balance, thus warding off the pathology which results from damming up the circulation and producing increased tension, and preserving corneal, vitreous and lenticular transparency.

As to the origin of the lymphatic system, it is the same proposition as the origin of the vascular system. It is believed that the lymphatics originate from the veins and that they, like the blood capillaries, grow thru proliferation of their own tissue. They form a system of absorbents that regulates the quantity and quality of tissue liquid, and, according to L. M. Kuyume, of Tokio, they play a great part in regulating the constitution of the blood. In such organs as the muscle and ductless glands, increased circulation associated with functional activity is always accompanied by an accelerated flow of lymph. The disposal of waste products is also a function of this system. Certain authorities look upon the lymphatic system as an emergency mechanism, as when it is called into action in the presence of severe septic accidents.

The reason given by anatomists for the failure to demonstrate satisfactorily the lymph channel of ocular structures, is the valves which they contain, which make the injection of fluids from the center out extremely difficult and up to date impossible, also their copious anastamosis and extreme minuteness. However, M. Leboucq claims that he has demonstrated an intraocular lymph circulation. Details of his experiments cannot be given in a paper of this character. His demonstration of the existence of lymphatics in the orbit seems quite conclusive, as follows:

When the specimen is fixed, the contents of the orbits are eviscerated and treated in the usual manner. Under this treatment, all nourishing fluids are held in place; the veins and arteries of the orbit are filled with coagulated blood; the smallest capillaries are recognizable by the presence of the formed elements of the blood. Besides the bloodvessels, the lymph vessels are plainly visible, distended by lymph and

lined by their characteristic endothelium. Such lymph vessels are found all along the principal veins.

Most interesting are some experiments recently made by S. A. Petroff, of the Research and Clinical Laboratory of the Trudeau Foundation, in attempting to work out an immunizing proposition in regard to tuberculosis. A series of fifty guinea pigs were sensitized with dead tubercle bacilli. He made an ocular injection of living virulent organisms directly thru the center of the lens into the vitreous humor, after which the animals were killed and histologic examination made in regard to the neighboring lymph nodes. Some of the organisms were carried to the auricular lymphatics and others to the cervical, and some reached the bronchial nodes, thus demonstrating very clearly that both the anterior and vitreous chambers are part of the lymphatic system.

You are all familiar with the splendid work done by F. Park Lewis, of Buffalo, in the study of the vitreous body. He has called our attention to the fact, that when a fresh eyeball is opened, a watery fluid is immediately discharged. This he calls vitreous aqueous, and pronounces it lymphoid secretion, claiming that it is continuous with the intraocular lymph spaces connecting the optic nerve sheath and the postlenticular spaces.

M. Uribe Troncoso has recently demonstrated that Schlemm's canal is normally a lymph space. The result of his findings were published in the A. J. O., May, 1921. He referred to the work of Schwalbe, who first demonstrated that this canal belonged to the lymphatic system, but later on changed his mind.

Troncoso describes Schlemm's canal as a plexiform organ, differing from veins by the complete absence of proper wall, the canal and tubes being formed only by an endothelial layer enclosed between the scleral fibers.

Most oculists I think agree with Dr. M. Uribe Troncoso, that by direct examination of the region of the angle, heretofore entirely concealed to our own eyes, the problem of the true nature of Schemm's canal, already solved by physiologic research, has been settled, and its lymphatic character asserted beyond doubt.

Dr. Birch-Hirschfeld also claims to have demonstrated a lymphatic apparatus in the orbit. Lymphangiomas, which occasionally are found in the orbit, indicate the presence of lymphoid tissue in this locality.

Treacher Collins declares that there is some uncertainty as to the nature of the lymphatic system of the orbit of man. Most authorities agree now that Tenon's capsule is in no way connected with the lymphatic system. There is no doubt as to the conjunctiva containing lymphatic vessels, which form themselves into a superficial and deep plexus.

Collins stated that perineural lymph channels surround the corneal nerves, both in the substantia propia and where they pass forward thru Bowman's membrane to the epithelium. Likewise, the lids contain deep and superficial branches, the former draining the skin and the orbicularis muscle, and the latter the tarsal plate. This system alsc connects with the preauricular lymphatic gland and the submaxillary gland.

I quote this on account of the high authority. Lymph vessels never have been demonstrated in cornea, lens or vitreous bodies, nor should we expect to find them in a stable optic apparatus.

Dr. Harold Gifford, in a very elaborate paper recording the results of many experiments, published in Knapp's Archives, 1892, declares that the corneal lymph stream, if one exists, is too weak to affect perceptibly the diffusion of fluorescein or the progress of pigment articles thru its tissue.

Some anatomists teach, that wherever there are blood vessels, there are lymphatics. They cannot always be demonstrated, but they are there.

Dr. Charles R. Bridgett, of the University of Pennsylvania, in a recent very exhaustive paper on "Sclerosis of the Central Artery of the Retina," published in Volume IX, A. J. O., declares that all vessels are plentifully supplied with lymph channels, which, in the main, run in the adventitial coat and the perivascular stroma. Now it is an accepted fact, that bacteria and foreign particles in general are commonly conveyed by way of the lymphatic channels, and it seems very likely that in such manner periarterial reactions may arise.

The therapeutist utilizes the lymphatic circulation to overcome many of the most serious pathologic conditions that the ophthalmologist is called upon to treat.

According to J. Herbert Fisher, the suprachoroidal lymph spaces communicate by lymph channels along the vorticose veins with Tenon's capsule, and thus drain into the intervaginal spaces of the optic nerve. Also, in the embryo the

main lymph space of the vitreous is the canal of Cloquet, and the main lymph flow must be from the periphery centripetally to the canal of Cloquet. He believes this posterior flow of waste products of the vitreous continues after birth and after the ciliary crypts begin to function and the anterior chamber contains aqueous. In certain low grade vitreous infections after operations, Fisher has found clinical evidences of lymph circulation thru the vitreous canal posteriorly.

I think we will all agree with Fisher, that if we knew more about the circulation of intraocular fluids, we should feel more confidence in some of our ocular therapeutics. He speaks in the highest terms of the use of urotropin as a most reliable remedy in the treatment of toxic cyclitis. This is administered intravenously, and is conveyed quickly into the anterior chamber to the lymphatic routes in the form of formaldehyd.

Similarly good results are recorded in von Graefe's Arch. für Ophthalmologie by Lutz, who reported a large number of cases of septic inflammation of various parts of the eye resulting from a septic focus which were quickly and satisfactorily relieved by the above named therapeutic agent.

Fisher's observations on the beneficial effect of intravenous injections of urotropin in low grade vitreous infections where there was little evidence of anterior involvement, suggest the posteriorward flow of lymph thru the vitreous toward the disc, and possibly the filtration possibilities of the lamina cribrosa in a manner similar to that of the spaces of Fontana.

Cushing uses urotropin as a prophylactic against sepsis, preliminary to operating on the pituitary body by the transnasal route, thus utilizing the lymphatics which are richly supplied in his field of operation.

Knowledge of intraocular lymph circulation should be essential for successful surgical procedures, but is complicated and most of the text books help us but little. A very clear and satisfactory tracing of the intraocular circulation will be found in Parsons' Diseases of the Eye.

For the rhinologists, the anatomists have worked out the lymphatics much more thoroly than they have for the oculists. The lymphatic system of the nose and adjacent sinuses is quite well understood. Altho, strange to say, according to Barnes, no afferent lymphatic stems have been demonstrated leading to the tonsils, the efferent have been clearly demonstrated.

Prof. J. Parson Schaeffer, in a recent paper, after referring to the very thin and filmy structure which separates the sphenoid sinus from the optic nerve, comments upon the obscurity of the applied anatomy in this region which now prevails, and urges the need of a more detailed study of the lymphatic drainage of the accessory nasal sinuses and tissue spaces about the optic nerve and commissure.

The lymph nodes play a most important function in this system. Cohnheim appreciated their importance, by claiming that the source of a given infection can be detected if the sentinel lymphatic gland first showing enlargement can be located. This brings to our mind the enlargement of the lymphatic gland in front of the ear, which normally is not visible but becomes enlarged as the result of a number of acute infections of the conjunctiva, and the enlargement of similar glands in the neck and at the angle of the inferior maxilla as a result of luetic and tubercular pathology of the eye.

Toxic agents are commonly distributed in three ways: thru the blood, which is rapid; thru the lymphatics, which is slower; and by local extension from pathologic lesions.

It is by way of lymphatics that we are chiefly interested in this paper. It is claimed that when a pathologic septic focus takes place in the body, it is the result of a temporary breakdown of the lymphatics, thus indicating the importance of comprehending this system of circulation and utilizing it for a therapeutic purpose.

Dr. E. L. Jones, of Cumberland, has repeatedly called our attention to the importance of this system in treating septic conditions, by bringing about the maximum physiologic lymphatic reaction by the use of subconjunctival injections of mercury oxycyanid. He looks upon lymph as Nature's antiseptic and tissue restorer, and claims that it goes to every part of the eye that a pathologic germ can reach. Many of our best known and most reliable ophthalmologic workers utilize some therapeutic agents for stimulating lymphatic activity in overcoming lenticular, vitreous and corneal opacities, as well as for absorbing hemorrhages and restoring loss of tissue structure.

Dr. J. B. Kirby, at our last meeting read a most stimulating paper, "A Study of the Nutrition of the Crystalline Lens", in which he referred to an unknown substance, acting perhaps

in a specific manner, which maintains the lens in a normal condition thruout life. May not this be the highly specialized protein of the lymphatics acting in harmony with one of the glands of the endocrin system, having the function to stimulate and inhibit?

It has been demonstrated that cataractous lenses and those of old people are deficient in protein. Have we not here a strong hint for rational therapeutics? But the secret of permanent lens transparency is still a mystery waiting for the faithful research worker to solve.

We all agree with Dr. Lancaster, I know, in saying that Nature stands ready to give up her secrets to the understanding mind.

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DISCUSSION.

Dr. W. W. Lewis, St. Paul, Minn.: The lymphatic system has always seemed to me a rather neglected study, one very unsatisfactorily disposed of. On the other hand, some of the stumbling blocks that have been interposed to the acceptance of sound, rational theories seem to me to be without warrant or good reason.

I think we have enough evidence to believe that the lymphatic system is a closed system. But it is a one way, efferent, closed system, which

returns the tissue fluids to the center of the animal organism for redistribution, after it has served as a medium of interchange of nutrition to and waste from the tissues, and, too, after its filtration, purification and disinfection at the lymphatic glandular depots en route.

I think there can be no doubt that the tissue fluid lymph originates from blood plasma, and that after diffusion outwardly from the arterial capillaries, it is conveyed by a closed system, made up respectively of tissue spaces, lymph channels and lymph vessels. Its course can well be pictured in one's mind as the course of a stream of water thru a meadow swamp or morass. A definite closed circulation may be just as difficult to trace in the swamp as in the tissue spaces, but nevertheless it is there, and the choroidal circulation is a good illustration of such a spread out, closed circulation in the animal organism.

The argument that tissue fluids of different chemical makeup prove dissimilarity in origin, I think is without good reasoning. Nutritive demands of the different tissues may cause a selective difference in lymph chemistry, and activities of the different tissues may cause a great difference in waste output contained in neighboring lymph. We have a good example of this in the lactic acid content in lymph from muscular regions.

We know that some glands of similar anatomic and histologic makeup have altogether different secretory products. Why may not lymph from tissues of different activities also be of different chemical makeup?

In ophthalmology, the lymphatics afford a big field for further study. While definite lymph channels may be absent in the orbit, the liberal distribution of lymph cellular elements, as demonstrated by their staining properties, would indicate considerable lymph tissue present, and, as pointed out by Doctor Fulton, the frequency of lymphomatous tumors in the orbit is significant. The same evidence by staining is present intrabulbarly in the uveal tissues.

The theory that lymph is a secretory product of the endothelium is far fetched. It seems to me most satisfactory to believe that blood plasma, lymph and tissue fluids are one and the same, but modified by the different cellular activities and local demands, and conditions existing to change their chemical makeup.

The lymph circulation as a medium of therapy is most interesting, and I know it is of chief interest and incentive to Doctor Fulton in connection with his nonsurgical cataract work.

The intraocular lymph, I believe, can be considered in a like manner to the general systemic lymph. Its origin from blood plasma contained in the uveal structures and its retention in the globe under a delicately balanced mechanism controlling income and exit, serve the purpose of pressure maintenance as well as that of a carrier of nutrition and waste. Intraocular therapy thru the blood and lymph circulation seems, therefore, altogether rational, whether administered locally or constitutionally.

The long enduring discussion of intraocular lymph and its avenues of circulation and exit from the globe seems to be gradually coming to indicate, that what formerly appeared very divergent views, will prove to be all partly right, and that all lymph like fluids in and about the globe, including Tenon's capsule and spaces, are the same tissue fluid modified only by the body chemistry to meet the differing local requirements of physiology.

IRIDENCLEISIS WITH WATER TIGHT CLOSURE OF THE CONJUNCTIVA.

S. R. GIFFORD, M.D.

OMAHA, NEBRASKA.

I have two objects in presenting this subject for your discussion; first, to bring out certain advantages of the iris inclusion operations in chronic simple glaucoma, especially as regards safety, and second, to call attention to a form of conjunctival suture which I have found to be of real value in any operation where a filtering scar is desired. The iris inclusion operations were first brought to general attention by several papers of Holth in 1906 and 1907,[1,2] altho Herbert[3] and Bader[4] had previously produced iris prolapses in their iridectomies and appreciated their value. Holth observed several cases in which a correctly performed iridectomy on one eye had failed to reduce tension, while in the fellow eye, where an accidental prolapse of iris had occurred, the tension remained low. He was successful in reducing tension in eyes where iridectomy had failed, by a second operation in which a piece of iris was left under a conjunctival flap. He then operated on 21 eyes, producing iris inclusion with iridectomy or iridotomy performed in various ways, with 85% of successes in reducing tension. He showed, in 1907, sections of an eye operated upon successfully with iris inclusion, but lost by an accident three months later, in which a fistula lined with pigment epithelium from the iris could be seen under the conjunctiva. He has recently[5] shown sections of six such eyes, removed after death, which all showed a similar mechanism of subconjunctival filtration. Collins[6] had previously stated that all the cystoid scars after iridectomy which he had examined histologically were shown to be due to inclusion of iris tissue. Besso[7] has recently examined sections of numerous rabbits' eyes on which a Lagrange operation had been performed, and found that the only eyes showing any filtering thru the sclerectomy opening were those in which an iris prolapse had occurred. Borthen,[8] in 1909 and 1911, reported a modification of Holth's

operation, the iris being drawn out under the flap, but not incised. He believed the good results of this procedure were due to traction on the lower half of the iris, the chamber angle being thus opened up for drainage, and hence proposed the name iridotasis. This has been the name adopted most widely in America, following the report on a small series of cases by Roy in 1916.[9] Holth used the name iridencleisis for his operation, and since Borthen's idea of the mechanism of the drainage produced has been generally abandoned, it would seem proper to use the name "iridencleisis" for any iris inclusion operation and "iridencleisis with meridional or basal iridotomy" according to which of these procedures is employed. What Verhoeff[10] described as a modified iridotasis, is very like Holth's iridencleisis with basal iridotomy, and since the sphincter is left in place, at least according to his most recent description, could hardly act as an "iridotasis" in Borthen's sense. Verhoeff mentions its advantages in buphthalmos, a point also emphasized by Holth, and also uses it in chronic congestive glaucoma. Galetski-Olin,[11] reporting 285 operations of various types for simple glaucoma, concludes that Borthen's technic is technically the simplest and attended with fewest complications. The tension became normal in 90% of his cases, and central vision was better preserved than by the other operations. Wilder[12] uses Borthen's technic, without iridotomy, and reports 48 operations, 10 for acute and 38 for chronic glaucoma, with tension successfully reduced in all but one case. Goldenburg[13] also uses this technic, and reports good results with very few complications. He does not mention the number of cases operated upon. Gjessing[14] employs iridencleisis with meridional iridotomy, and reports on 92 operations with vision the same or better and tension normal in 83.7%, 8 months to 11 years after operation. Tension was not below 12 mm. (Schiötz) in any case, which he emphasizes as a point in favor of the operation. He believes the operation is the least dangerous of any in chronic glaucoma. Ikonomopoulos[15] and Pillat[16] also use the Holth technic, the latter having recently reported on 101 operations, with 85% of good results.

The technic I have used is much like that employed by Holth, except for the conjunctival flap. Eserin is not employed, but 2% butyn drops are used instead of cocain to avoid dilation of the pupil. Injection of two drops of 2%

cocain over the superior rectus gives sufficient anesthesia for fixation and iridotomy. The superior rectus is seized with toothed forceps provided with a catch, as far back as possible, at least 1 centimeter from the limbus. A straight horizontal cut is made in the conjunctiva, just below the forceps, extending out as far as the border of the cornea on each side, and the conjunctiva is dissected up as far as the limbus, but not onto the cornea as for a trephining. Fixing the eye by the toothed forceps, while an assistant holds the flap up, a fairly narrow keratome is inserted in the sclera 1½ mm. back of the limbus and introduced into the anterior chamber, mak-

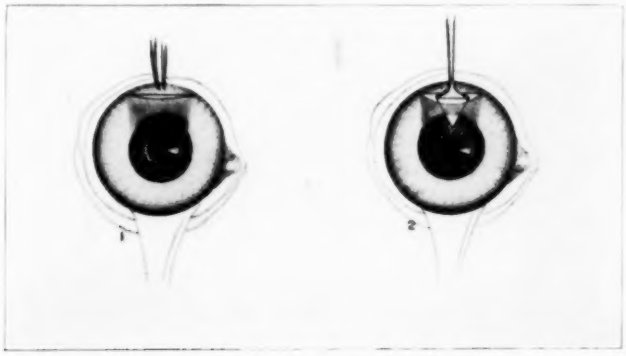

Fig. 1. Fixation on superior rectus. Straight conjunctival incision undermined to limbus. Fig. 2. 4 mm. scleral incision. (Fixation forceps not shown.)

ing an incision 4 mm. long. The iris is drawn out by iris forceps, and a vertical cut made thru the sphincter and iris. One pillar of the iris is replaced by massage or by a spatula, the other being patted down under the conjunctival flap. The conjunctival incision is closed by a running suture of silk, the stitches being placed fairly close together and extending well beyond both ends of the incision. The stitches should be placed from above, to avoid touching the cornea with the needle holder and so reopening the anterior chamber. No knots are used, about 1 centimeter of thread being left loose at each end of the suture, after it is pulled tight. Usually only a few drops of aqueous are lost, and there is nearly always some anterior chamber present at the close of the operation. The dressing is changed after 24 hours, and the stitch is removed after six days. The tension is taken after six days, and if it is above 12 (Schiötz), gentle massage is begun, or this may be begun sooner in certain cases.

Results: The technic described above has been carried out on 21 eyes, all but 2 operations being for chronic simple glaucoma, and the other 2 for buphthalmos. The time elapsing since the operation varies from 2 years to five months, 3 being too recent for anything but the immediate operative result to be estimated. The last recorded tension in each of these eyes was 11, 24, 10, 9, 3, 15, 15, 17, 20, 11, 18, 20, 18, 24, 25, 11, 14, 12, 17, and two not recorded. That is, no tension was above normal, and only one was below 9 (Schiötz). In three eyes, the tension gradually rose somewhat above normal, reaching 36, 35, and 32, and in these eyes a subconjunctival prolapse puncture

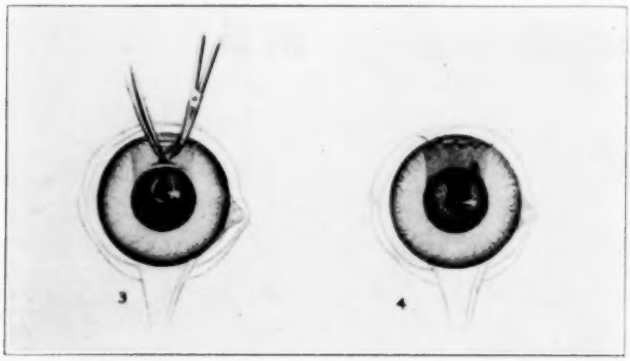

Fig. 3. Vertical cut in sphincter and iris. Fig. 4. One pillar of iris everted and left out under conjunctival flap. Conjunctiva closed with running suture. (Pillar held by forceps should be the one left under flap and not as shown in picture.)

was performed, resulting in tensions of 15, 18, and 25 at last examinations. This procedure, as described by H. Gifford,[17] is carried out under local anesthesia in the same way as an anterior sclerotomy, except that the Knapp knife or thin cataract knife is introduced well back thru the bleb, under an area of additional edema made by injecting cocain, and passed thru the prolapsed iris into the anterior chamber. It is followed by massage, and is used with the idea that pigment epithelium will grow out along the knife track, allowing freer filtration. It often reduces tension permanently in eyes where not quite enough effect was obtained by a sclerectomy or iridencleisis.

Vision before operation varied from 20/20 to counting fingers and, except in two cases, remained practically the same after operation. The fields, except in these 2 cases,

which were taken at the last examination showed no appreciable loss during the period of observation. In one of the cases mentioned above, the chamber was full 24 hours after operation, but after 48 hours, the stitch was pulled in removing the dressing, the chamber emptied, and remained empty four days. The field was very small, and became smaller after operation, central vision falling from 20/50— to 4/200. This contraction of the field with loss of central vision is something which occurs after trephining, and is the complication most to be dreaded when the field is close to the fixation point.

Fig. 5. Dotted line indicates diffuse area of conjunctival edema after iridencleisis. No localization of bleb in this case.

I believe the rarity of its occurrence after iridencleisis with running suture, is due to the fact that the chamber fills early, and in fact is seldom emptied, so that there is no sudden and prolonged hypotony. There were a number of cases with small fields where this was feared, in which this relative factor of safety in the operation was apparently demonstrated. Mr. F., had fields within 15 degrees all around in one eye, and within 30 in the other, and suffered no loss of vision immediately after his operation. His fields are still the same after 20 months, altho the vision in one eye gradually declined from 20/100—1 to 15/200. This was one of the cases

in which the tension rose to 35, requiring two punctures to reduce it, and the loss of vision was ascribed to this return of tension. Everett O., the case of buphthalmos, had fields within 15 degrees in each eye, and these remained the same two years after operation, vision being also the same, 20/100 and 20/30. Except for the one case in which the chamber emptied, there were no complications of any significance for vision. Several had slight bleeding from the iris, which cleared up rapidly. There were no vitreous losses, no injuries to the lens, and in none was the postoperative reaction serious enough to be called an iritis.

One complication occurred which should be mentioned, altho it has had no effect on vision. In the case of buphthalmos previously mentioned, a staphyloma appeared at the site of the scleral incision in the poorer eye. This has gradually grown somewhat larger, reaching the size of a pea, and consists of uveal tissue well covered by conjunctiva. The tension is 25, as opposed to 11 in the other eye, but vision has remained the same as before operation, 20/100. Altho no difference was noted at the time, I believe the incision must have been too far back, and for this reason I try not to make it further than 1½ mm. from the limbus, and not 2 mm. as advised by Holth. The same thing occurred in another case, not included in this series because iridotomy was not performed, and I believe the possibility of such a complication developing is a reason for prefering iridencleisis with iridotomy to Borthen's technic. In this case vision has also remained the same, 20/50, and tension is 25.

Dr. Harold Gifford's[17] series of 125 operations, all but the last 15 of which were done without the running suture, included very few complications, except those which I believe were due to hypotony. In most cases the chamber was empty at the first dressing, requiring four to ten days to fill. In three the chamber remained empty as long as the patient was observed (2 years in one case), and in all of these cataract developed. In two others, where the chamber remained empty no longer than usual, there was further loss of an already small field, the fixation point becoming involved. In several cases, subsequent prolapse puncture was performed, and from the records, only two patients are seen to have a tension above 30 (Schiötz) at the last examination, altho tension may have recurred in others who could not be followed.

When compared with ordinary iridectomy, the advantages of iridencleisis in chronic simple glaucoma are well known. A much larger percentage of iris inclusion operations produces a permanently lowered tension, so that iridectomy is reserved by most operators for acute or secondary glaucoma. With the newer modification of iridectomy, as described by Török and Suker, I have had no experience, but I believe that for most cases of chronic glaucoma, some operation which will produce a filtering scar is necessary.

Any operator who performs a sclerectomy, whether according to Elliot or Lagrange, will admit that the operation must be exactly performed in order to be effective, and that it is far from a simple operation to perform correctly. The ciliary body may be injured by a cut placed too far back, or the conjunctival flap buttonholed by one placed too far forwards. Vitreous may even be lost by a trephine which is not carefully handled, or the lens may be injured. While these should be rare complications in the hands of experienced operators, they have probably caused a good deal of trouble to those doing their first few operations, or to those who do not perform glaucoma operations frequently. Certainly, by the technic described above, such complications should be exceedingly rare. The only possibility of injuring the ciliary body would be by an incision placed too far back, and if a distance of 1½ mm. from the limbus is observed and the keratome directed well forward, this complication could hardly occur, and has not in my experience. I have buttonholed only one conjunctival flap, and in this case the perforation was very small and healed, so that the chamber was refilled in 24 hours. I have never seen a vitreous loss in my series, nor in about 129 iridencleises performed by Dr. Harold Gifford, whereas this occurred 7 times in 405 trephinings reported from Moorfields Hospital by Davenport,[18] nor have I seen the lens injured at the time of operation.

Even when perfectly performed, the operation of sclerectomy has inherent in it certain dangers to the future of the eye. Chief of these is the dreaded late infection. This occurred in 14 cases in Davenport's series, and Wessely,[19] from available reports, gives 2% as the average incidence of late infection after trephining. While not always severe, only two of Davenport's cases requiring evisceration, for example, it is always dreaded, and this dread is largely responsible for the

many modifications of the Elliot and Lagrange technic, and for the newer methods of iridectomy. It was this same fear of leaving a path for infection into the eye that deterred many operators from trying the iris inclusion operations, but this fear has received very little support from the facts. Holth, Borthen, Wilder, Goldenburg, Ikonomopoulous, Gjessing, and Pillat report no late infections in their series. Among Dr. Harold Gifford's cases, a slight superficial infection of the bleb occurred twice, but was easily controlled. This relative freedom from infection is apparently due to the usual avoidance of the thin, prominent, avascular blebs often seen after sclerectomy. Since the conjunctival flap is not carried down onto the cornea, no place of least resistance is found, such as the thin corneal edge of the flap in a trephining. Often no definite bleb is formed, but a large area of conjunctival edema, which pits on pressure. (See Fig. 5.) Gjessing found Seidel's fluorescein test for conjunctival fistulization to be never positive in a properly performed iridencleisis, while 50% of trephinings showed fistulization at this thin area over the limbus.

Another serious danger, not so generally thought of, is that of prolonged hypotony. While this occurs less frequently after iridencleisis, according to Wessely and Gjessing, who found no tension below 12 (Schiötz) in his 76 cases, it does occur. Wilder mentions four cases, and I have seen several among Dr. Harold Gifford's cases, in three of which the chamber never reformed. The obvious danger of a prolonged absence of anterior chamber is that the lens will become opaque. Hepburn[20] saw this occur two or three times, and Knapp[21] three times out of six eyes with prolonged hypotony after trephining. Besides this, one of the eyes went on to pthisis bulbi. The rapid loss of field, sometimes involving the fixation point, apparently is due to sudden reduction of tension, and should occur less often if the hypotony is less marked or prolonged. This is also undoubtedly true of choroidal detachment, which is due to the hypotony.

The use of the water tight suture described above is, I believe, of the greatest value in hastening a reforming of the anterior chamber, and thus in preventing later hypotony. It was described by R. Foster Moore[22] in 1923, for use in trephinings, to avoid the prominent cystoid blebs so often seen when the flap was made according to Elliot. I first saw Brewerton use it in trephinings, and have used it since in iridencleisis for the following reasons:

1. Early reforming of the chamber, with avoidance of hypotony. Moore says the chamber was sometimes reformed eight hours after operation, and I have always seen it reformed after twenty-four hours.

2. Conversion of the whole flap into a cushion under which filtration occurs. The reforming of the chamber occurs early, not by closure of the scleral wound, but by closure of the conjunctival wound, the whole flap being raised up by aqueous. This avoids the dangerous blebs which occur when only the thinnest part of the conjunctiva is raised up, the rest being adherent to the sclera.

3. Better reduction of tension. Apparently the lips of the wound are kept from healing so early and firmly by this slight pressure under which the aqueous is held.

These advantages of the water tight suture in iridencleisis apply equally well to any form of filtering operation, especially to trephining, in which hypotony and thin avascular blebs are more apt to occur. In Davenport's series of 405 trephinings, this suture was used, and hypotony is not mentioned as among the complications which occurred. The only disadvantage is the extra two or three minutes consumed in making a careful suture. Here fixation on the superior rectus is a great convenience. I have used this suture in trephinings and iridectomies, and believe that the extra time consumed is more than repaid by its advantages. In the first 20 operations, the chamber was always refilled after 24 hours, and it seemed possible that hypotony could be absolutely prevented by a properly placed suture. In the last case, however, altho the suture was made as usual, the chamber was very shallow at the first dressing, and filled slowly, being more shallow than normal after a week. In this operation, the chamber emptied completely, which can, in a majority of cases, be avoided, and this suggests that there may be eyes with a weak zonula or some other anatomic peculiarity, in which, when the chamber is emptied, the whole lens system comes forward, and is not replaced until a pressure in the anterior chamber is developed greater than that required in most eyes.

Summary.

1. Iridencleisis with meridional iridotomy is a simpler procedure than sclerectomy, and one less liable to complications caused by slight errors in technic.

2. It is effective in reducing tension permanently in the large majority of cases.

3. It is of value chiefly in chronic simple glaucoma and buphthalmos.

4. It is not recommended in absolute glaucoma, acute glaucoma or secondary glaucoma where some iritis may still be present.

5. While there have been no failures to reduce tension materially by this operation, there is possibly a greater tendency for tension to rise slightly above normal than after successful sclerectomy. In these cases, subconjunctival prolapse puncture is of great value. Where it is known that a case can not be followed for several months after operation, sclerectomy might offer a better chance of success than iridencleisis. Gjessing notes that tension is sometimes reduced slowly after this operation, and he often uses miotics for a time until it remains normal without them. I have found massage of more value than miotics, especially in the first few weeks after the operation.

6. It involves less danger of late infection than sclerectomy.

7. When combined with the water tight suture, a safer, more effective bleb is formed and hypotony is not apt to occur.

8. For this reason, this procedure is of especial value in cases with fields cut down to near the fixation point.

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DISCUSSION.

Dr. Edward Stieren, Pittsburgh, Pa.: My attention was called to this operation by the papers of Dr. Dunbar Roy and Dr. David Harrow which appeared about ten years ago. Having had, as we all have, unsatisfactory results from iridectomy and trephining, the simplicity of the operation, and the good results of the American operators appealed to me. Several years later, at the American Ophthalmological Society at New London, I reported six cases with good results. Since then, I have operated on 22 eyes, with equally good results. I have not found it necessary to deviate from Borthen's original operation. Dr. Gifford's modification seems to me only to complicate the operation and make it harder to perform. The original Borthen consisted of a corneal section well back in the limbus, making a 4 or 5 mm. curved conjunctival flap. Before the operation, a drop of 1 per cent. atropin was instilled into the eye. The iris was grasped so that it came out with the under surface on top. But I do not think now that such a large conjunctival flap is necessary; two to three mm. is enough, but it should contain all the conjunctival tissue.

To me, the good results from this operation are not so much due to the filtering scar as to the drawing away of the iris from the filtration angle below. Almost uniformly, the conjunctiva is perfectly flat above: it does not seem to make any difference whether there is a bleb formation or not, and the results are immediate and permanent, the tension is reduced, and the subjective symptoms improve or disappear.

It seems to me, that by making the incision as low as Dr. Gifford does, in event of cataract forming afterwards, the cataract extraction would be much more difficult to perform than if the incision were in the limbus. I have had on three occasions to remove cataracts after this operation, and by placing the corneal incision two or three mm. forward, the extraction of the cataract was not at all difficult.

After all, the choice of operation depends upon the results we get, and I know that if I were to have a simple progressive glaucoma which miotics could not control, I would want Dr. Harrow, Dr. Roy, Dr. Wilder or Dr. Gifford to do an iridencleisis on me.

Dr. James M. Patton, Omaha, Neb.: In discussing Dr. Gifford's paper, I wish briefly to supplement the report of two cases of buphthalmos which I made two years ago at Chicago. The first case was that of a child with typical buphthalmos, increased tension, large cornea, and reduced vision, in which I did an induced conjunctival prolapse, which is not a typical iridotasis but amounts to the same thing. This child subsequently developed a staphyloma as large as the end of my finger, which after several punctures and a resection, finally ruptured with secondary shrinkage of the globe. The child was defective mentally and does not see so much worse than before the operation. So far as the operation is concerned, it was a failure.

The other was a case of juvenile glaucoma in the right eye, in a boy of fifteen years. The tension was about 70 when we first examined him, but came down to 37 in each eye under eserin. An iridotasis was done on the right eye and a peripheral iridotomy (Curran) on the left, resulting in normal tension in each eye. He disappeared for a couple of

years, and has just returned with a tension of 17 in the eye with the iridotasis and 37 in the other. An iridotasis was done on the left eye and has reduced the tension, but it is too soon to tell what the final outcome will be.

In cases of buphthalmos, I am inclined to think that iridotasis offers the safest means of decompression at our disposal, but the possibility of an enlarged staphylomatous bleb must be considered.

Dr. S. R. Gifford, Omaha, Neb. (closing): I am glad Dr. Patton mentioned the cases of buphthalmos, because it seems to me in these cases particularly it would be interesting to obtain a series of reports of results by this operation, which I believe is safer than trephining in just these cases, where we are likely to lose vitreous by trephining.

I especially wished to emphasize the advantage of this running suture in iridencleisis, trephining or any other filtering operation. It was described by Foster Moore in 1919, and since 1921 I have been using it for three reasons: First, because we avoid the thin edge of the flap seen so often when we dissect the flap into the limbus, which is the point of danger from late infection. The conjunctiva closes before the scleral wound, leaving a conjunctival flap which is uniformly edematous and has fluid under it. Second, I believe we get more effect in reducing tension from the iridencleisis, as the scleral wound seems to be held open longer. Third, we avoid hypotony. The chamber was filled in 24 hours in all but one of my cases, which was the exception by the technic I had seen used before.

I would not have had the temerity to present this small series of cases, had I not assisted my father in doing about 125 operations before that, and seen proof in cases, some of which have been watched seven or eight years, that iridencleisis can keep the tension down apparently permamently.

With all respect to Dr. Stieren, I believe that Borthen has not proved his theory, that the effect is due to opening the opposite chamber angle, by the examination of eyes removed after successful iridotasis. Holth has furnished proof that iridencleisis causes a filtering scar. On account of the cases which developed a staphyloma where the iris was not incised, I believe it is safer to incise it and turn back a good piece.

SOME CHANGES AT THE POSTERIOR POLE FOLLOWING CONTUSION INJURIES OF THE EYE.

HAROLD J. ROTHSCHILD, M.D.

ST. PAUL, MINNESOTA.

Contusion injuries of the eye seen in civil practice are commonly caused by the impact of blunt objects, such as pieces of wood or iron, B.B. shot, corks, stones, tennis balls, a fist, etc., striking the anterior part of the eye.

In addition to more or less trauma to other parts of the globe, they not infrequently cause concussion changes to occur at the posterior pole, such as commotio retinae, retinal and choroidal hemorrhages, retinal detachments and rents, choroidal rupture, "hole" in the macula, and macular pigmentary changes.

While concussion disturbances do indeed occur thruout the fundus, we are constantly reminded of the preference of these lesions for the macular and perimacular regions.

The reasons for this preference are not entirely clear. Altho some investigators declare that contrecoup is an important factor in certain cases, it does not explain those instances where the macula is affected in spite of the site of contrecoup occurring at some other point. Other observers, among whom may be mentioned Haab[1] and Siegfried,[2] and more recently Lagrange[3] and Lister,[4] point out that this region is rendered more vulnerable to the sudden increase of intraocular pressure, because of its peculiar vascularity and greater delicacy of structure. With regard to indirect laceration of the choroid and retina, or of their vessels, de Schweinitz[5] states that, "they depend in part upon stretching of these membranes by the vibrations in the vitreous. They are frequently placed at the posterior pole of the eye and near the papilla. These situations are peculiarly liable according to some observers, probably because the sclera, thicker around the entrance of the optic nerve than elsewhere, does not readily stretch. Its resistance in this regard causes the effect of force to be more potent on the tissue just in front of it."

Others theories for choroidal rupture have at various times been advanced, such as invagination of the optic nervehead, fixing of the choroid to the sclera by the posterior ciliary vessels, forcing of the globe against Tenon's capsule and orbital contents, and stretching of the choroid when the optic nerve is suddenly made taut.

Traumatic affections of the macula lutea have been the subject of numerous studies. Among the best is one by Siegfried, who gives us the following summary of all cases observed at the University Eye Clinic at Zurich during a period of six years. There were 192 cases of contusion and concussion injuries where the fundus was visible. Of 167 observed shortly after the trauma, 138 cases, or 82.5 per cent, showed no changes in the fundus; 8 cases, or 4.7 per cent, showed a Berlin's opacity and a normal macula; 21 cases, or 12.5 per cent, revealed opacities both in the macula and in the outer part of the retina. In the last group of 21 cases, there were 16 where the opacities disappeared without permanent changes, the ultimate vision proving good or but little diminished. The remaining 5 cases had permanent pigmentary changes in the macula, which could be observed on the first, third, fourth, eighth, and nineteenth days; prognosis not so favorable. In 4 cases there developed an optic atrophy, and in 1 case, a change in the choroid; vision in the best instance was 6/60. Of the total number (192), there were 46 cases, or 24 per cent, in which there occurred permanent changes in the macula. Of these, a flying object was the cause in 22 cases, or 47.8 per cent; a blow as from a fist in 11 cases, or 23.9 per cent; an explosion in 4 cases, or 8.7 per cent; an arrow shot in 3 cases, or 6.5 per cent; a pushing trauma in 3 cases, or 6.5 per cent; a fall in 2 cases, or 4.3 per cent; cause unknown in 1 case, or 2.1 per cent. The prognosis is progressively worse when the injury is caused; (a) by flying objects and blows; (b) by pushing trauma; (c) by arrow shots; and (d) by explosions and falls. In conclusion, he states that macular changes have a tendency to increase with years, the choroid in this area becoming, probably, secondarily involved and explaining the whitish spots.

Leaving out of consideration those cases where the fundus is obscured by infiltrations of the media, it may be remarked that, in some instances, permanent lesions at the posterior pole are not visible for weeks after the injury. This may be

due to the tardiness in development of the secondary changes, for example the frequently late appearance of atrophic patches after choroidal hemorrhages. The prognosis should be guarded accordingly.

The purpose of this paper is to report four cases of posterior pole changes illustrated with new photographic color reproductions of water color sketches by the author.

Case 1. Choroidal Hemorrhages Followed by Chorioretinal Atrophy at the Posterior Pole: I. G., a boy aged 6, was injured August 31, 1926, when a stone shot from a sling struck his right eye. Arriving at his home two hours after the injury, I found the boy in much distress and with marked blepharospasm.

The left eye was apparently normal. Examination of the right eye revealed chemosis of the lids and bulbar conjunctiva, and cloudiness of the entire cornea. There was an erosion involving the inferior quadrant of the cornea and the adjacent few millimeters of conjunctiva. The anterior chamber, iris and lens showed no gross change, and the pupil was small. A dulled red reflex was obtained with the ophthalmoscope. Vision of this eye was reduced to hand movements.

September 3rd., the cornea was cloudy, the abrasion healing satisfactorily, anterior chamber normal. The pupil was in atropin mydriasis and round, and the crystalline lens was clear and manifested no change in position. No vitreous opacities were noted. The fundus appeared edematous at the posterior pole, the details blurred, the papilla hyperemic. Tension, — 1. Vision of the right eye, fingers at 5 ft.

September 7th., the ciliary flush was still prominent, but the cornea was less cloudy. The ophthalmoscope revealed small dark bluish red blotches in the papillomacular region, which were difficult to distinguish in the blurred fundus. Their outlines were irregular and blurred, and retinal vessels passed over them. Tension, —1. Vision, fingers at 10 ft.

September 15th., there remained slight ciliary flush and corneal cloudiness, the latter more noticeable at the inferior quadrant. The fundus showed little change since the previous examination. Vision of the right eye was 15/100, left eye 20/20.

October 11th., the eye appeared, externally, normal; the media clear. The ophthalmoscope revealed pigmentary changes extending from the papilla temporalward and involv-

ing the macula. The affected area had the shape of an irregular broad band about the width of the papilla, and the changes for the most part consisted of pale patches and a heaping of pigment within them. The outlines were not sharply demarcated, and a few small retinal vessels were seen passing over the lesion. At the macula, an interesting change was noted: A fine, rather uniform stippling of pigment, occupying an area a little more than half the size of the papilla, was divided horizontally by a narrow band of the width of a retinal vein of the first branching, and of the color of the normal fundus. The margins of the stippled area were not sharply circumscribed. The papilla was beginning to pale, and the retinal vessels were normal. The vision of this eye is 18/100. Tests for scotoma are unsuccessful owing, probably, to the youth of this patient. The accompanying sketch was made at this time. (Fig. 1.)

August 17th. The lesions have somewhat altered their appearance since the sketch was made about eight months ago. The pale patches, above mentioned, are now whiter, and the pigment granules within show more general distribution. The margins are sharply circumscribed and reveal occasional narrow borders of black pigment. The uniform stippling at the macula has been broken up into irregular areas, revealing small depigmented spaces, and the narrow straight band has now become indented and branched, and shows still lighter spots here and there. At one point, a choroidal vessel is bared. Vision has improved to 20/65.

Case 2. Rupture of the Choroid at the Macula: M. L., male, age 28, suffered injuries of both eyes November 17, 1925, when a charge of powder, used in quarry blasting, prematurely exploded. He was in a squatting posture, facing the tunnel in which the charge had been placed, and receiving the force of the explosion on the face, forearms, knees, and legs, he was thrown backward and rendered unconscious. He was removed to a near by hospital at Little Falls, Minnesota, where I saw him the following day. At this time, he was in a state of partial shock, and the skin surfaces above named, especially the face, were swollen, peppered black with powder and the seat of superficial burns. My notes proceed as follows: Right eye, lids are black and swollen and the cilia are burned. The temporal two-thirds of the upper lid shows a horizontal wound extending thru its entire thickness, the

conjunctiva is injected and chemotic, and there is some mucopurulent secretion. A linear scleral wound is seen, beginning 3 mm. from the corneal limbus at 10 o'clock and extending temporalwards a distance of about 8 mm., corresponding to the wound in the upper lid. A piece of wire fuse, protruding slightly into the globe, had been removed from this defect. The cornea is very cloudy and its luster is diminished. The pupil is in mid dilation, atropin having been instilled, and is oval, being drawn in the direction of the scleral wound. No details of iris structure or crystalline lens are visible thru the cloudy cornea. With the opthalmoscope, only a faint red reflex can be obtained. X-ray shows no foreign bodies in the globe or orbit. Vision is reduced to the perception of light.

The left eye was somewhat less severely injured, but will not be considered in this paper, since no changes at the posterior pole occurred. I saw him again 20 days later, when he was brought to Saint Paul for further hospitalization.

At this time, December 7th., the cornea reveals a dense opacity of its inferior half, in the midst of which a depression bears evidence of a rather extensive necrosis. The upper half is less opaque, and thru it can be seen no gross alteration of the anterior chamber and iris. The pupil is in atropin mydriasis and round, and the crystalline shows a small roundish opacity in its extreme nasal portion. The ophthalmoscope reveals the presence of dust like infiltrations and many stringy hemorrhages in the vitreous, and thru the upper half of the cornea, a red reflex is seen but no fundus details. Tension (fingers) is slightly lowered. Vision; counts fingers at 2 ft.

January 6th. The conjunctival and ciliary injection is receding. An opacity of considerable density occupies the lower half of the cornea; the corneal ulcer has been covered by epithelium, leaving a facet. A few horizontal linear opacities are noted in the temporal portion of the lens, and the vitreous hemorrhages are resorbing, but the fundus details are still obscured. Vision; fingers at 20 ft.

May 18th. The ophthalmoscope reveals changes in the macular region: A pale yellowish streak bordered by a lightly pigmented zone occupies a site at, or near, the fovea. It is almost vertical, the upper extremity being slightly nearer the papilla. The light streak is about 1¼ disc diameters in length,

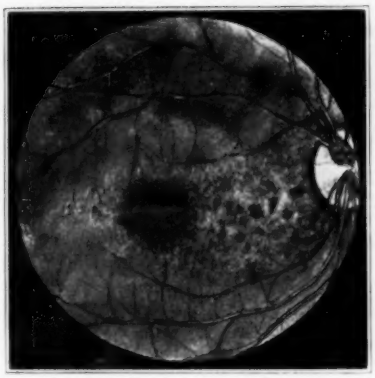
and about twice a retinal vein in breadth. The foveal reflex is not visible. Vision; 20/100. (Fig. 2.)

(While this case is strictly not one of contusion injury, but rather due to concussion, it seems that it may, with propriety, be reported here, for after all we are dealing with the concussioned fundus).

CASE 3. OLD RUPTURE OF THE CHOROID: Dr. C. A., a physician, aged 57, states, that when 25 years old, he was pulling a board from under a pile of wood, when it suddenly gave way and struck his left eye. He does not remember the subsequent course of the injury, except that for many years he had been vaguely aware of some slight loss of visual acuteness of this eye. A few years ago, during an examination of his refraction, his oculist noted a rupture of the choroid. Recently, I had the opportunity to observe this lesion, and was surprised to find that in spite of a rather long, old choroidal rupture between the papilla and macula, his acuteness of vision suffered none at all. The rupture extends outward and downward from a point above the papilla, following the curved temporal margin of the latter, and about one-half disc diameters therefrom, until it nears the level of the inferior margin, when it extends downward and slightly outward; it ends at a point between the papilla and macula, and somewhat below the level of the latter. The margins of the rupture are about one-third disc diameters apart, and are bordered here and there with clumps of pigment. Within the rupture, there is seen pale or whitish tissue intermixed with pigment, mostly in clumps. Retinal vessels pass undisturbed over the lesion. The papilla and retinal vessels are normal. Visual acuity, 20/20 with each eye.

Perimetric tests show no contraction of the visual field. With the stereocampimeter, a scotoma is outlined which corresponds rather closely to the form of the fundus lesion except that, above, it merges with the blind spot. The lower half of this scotoma is relative, and there are gaps in it. (Fig. 3.)

CASE 4. MACULAR "HOLE", PERIMACULAR CHORIORETINAL ATROPHY: J. C., male, age 37, when 12 years of age, fell on a stick of wood striking his left eye. Since the injury, the vision of this eye has remained markedly lowered. The ophthalmocope reveals at the macula a reddish tinted spot, about one-fourth the size of the papilla, and round. The outline is fairly sharp, except for a small part of its circumference below. At



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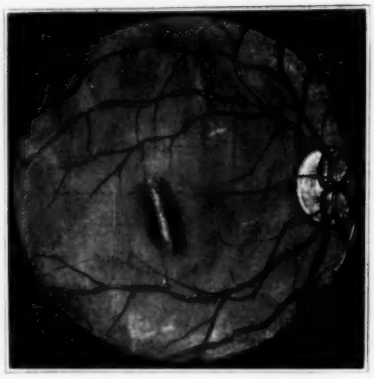

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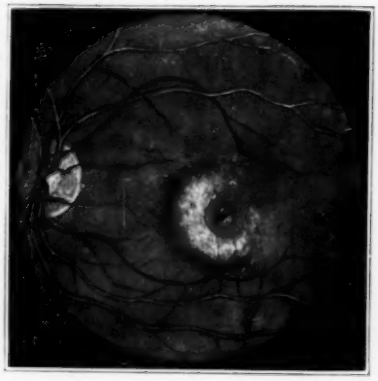
Case No. 3.



Case No. 4.

Fig. 1. Early stage of chorioretinal atrophy at the posterior pole, following a contusion injury of the right eye of a boy, aged 6 yrs. Vision, 18/100.

Fig. 2. Rupture of the choroid at the macula due to an explosion injury. Right eye of a man, aged 28. Vision, 20/100.

Fig. 3. Left eyeground of a man, aged 57. In spite of a rather extensive old choroidal rupture between the papilla and macula, the vision is 20/20.

Fig. 4. Left eyeground of a man, aged 37, showing a socalled "hole" in the macula with perimacular atrophy of 25 years standing. Vision: counts fingers at 5 feet.

various points in this area, small, decolorized spots are visible. Its level, as compared to the neighboring retina, shows no change. The color is not so deep, and the outline not so sharp, as those I have seen in more recent lesions. Surrounding its nasal circumference, and separated from it by a narrow margin of somewhat pale looking retina, is a crescentic whitish area of choriorentinal atrophy, about one-half papillary diameter wide, and this in turn is bordered by a zone of rather faint pigmentation. The temporal half of the papilla is pale. The retinal vessels are normal, and a few small branches can be seen coursing over the atrophic patch from below. The vision of this eye is reduced to the counting of fingers at 5 ft. An absolute central scotoma corresponds to the fundus lesion. This sketch is submitted in order to show the appearance of a macular hole 25 years after its inception. (Fig. 4.)

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DISCUSSION.

Dr. Arthur J. Bedell, Albany, N. Y.: The paper which Dr. Rothschild has read is timely, and surely brings to the mind of each of you some case that has caused you more or less annoyance. To emphasize certain points, slides are shown to illustrate the various changes that may take place in and about the macular region. These include the socalled colobomas, and a case of toxic retinitis, in which a true cyst developed in the macular region.

Because fundus photography is so new, it is essential that we learn to read a photograph. If the disc and macular region are included in the picture, then the larger light must be below, unless the patient has a high degree of myopia, which necessitates the use of one of the additional focusing lenses that are part of the camera, when we have a triplicate light and the middle image may be the largest.

At the meeting of the American Ophthalmological Society held in June of this year, I spoke about holes in the macular region and referred to some allied conditions. At that time, I drew attention to the fact that certain uveal tract diseases can, as stated by some other observers, produce a destruction of the retina in the macular region. In a case of uveitis with secondary increase of tension, which case could not be controlled by local measures, we did an iridectomy, and in the photograph, there is seen an arc of inflammatory exudate surrounding the macular region. It is,

therefore, quite necessary to always remember that we may have a macular hole without trauma.

Dr. Rothschild stressed the point that a prognosis should not be given immediately after an injury. This was well illustrated in a recent case, where a boy was struck over the forehead by a phonograph record. There were many hemorrhages thruout the retina, particularly in and about the macular region. By the comparison of a series of photographs taken at different times, the absorption of blood and the migration of pigment illustrated by Dr. Rothschild in one of his cases is clearly seen.

The types of traumatic detachment of retina in which the separation appears in the form of flat elevations is frequently overlooked. Chemosis of the retina of minor degree may simulate retinal detachment. In any case of detachment of retina, a destruction in the macular region may develop.

Cases of true traumatic hole in the macula are considered in the paper previously referred to, and the slides here shown are described in detail in that communication.

Dr. Rothschild's paper will surely arouse more interest in these cases and will prevent some disappointments if the prognosis is not given too early.

THE RELATIONSHIP OF INTRACRANIAL PRESSURE, CHOKED DISC, AND INTRAOCULAR TENSION.

ALFRED W. ADSON, M.D.,* and WALTER I. LILLIE, M.D.†

ROCHESTER, MINNESOTA.

Because ophthalmoscopic data vary in the different lesions of the brain and meninges, we have attempted by clinical investigation to establish the relationship of increased intracranial pressure, choked disc, optic atrophy and intraocular tension. This report is necessarily preliminary, as we have not yet had an opportunity to carry out all the investigations planned in the various types of intracranial lesion. By means of an apparatus devised for this purpose, it is possible to maintain and control intracranial pressure and drainage of the ventricles, and to record intracranial pressure continually for any period of time desired. During this period, we record simultaneously observations on the fundus, intraocular tension, changes in the field and neurologic phenomena, such as mental changes, convulsions, headache, vomiting and stupor, as well as pulse, blood pressure, and respiration.

It has been definitely established that the source of the cerebrospinal fluid is from the choroid plexuses of the brain, chiefly from the choroid plexus of the lateral ventricles. From the lateral ventricles, it flows thru the foramen of Monro into the third ventricle, then thru the aqueduct of Sylvius into the fourth ventricle, and thru the foramina of Magendie and Luschka into subarachnoid cisterns. The greater portion of the cerebrospinal fluid passes into the various cisterns, and is distributed over the surfaces of the cerebellum and cerebrum, then thru the villi of the arachnoid into the venous sinuses, the pacchionian bodies, the longitudinal sinus, and the communicating veins in the arachnoid and in the brain. The remaining portion is carried into the spinal canal, and is absorbed thru the lymph spaces about the spinal root and

*Section on Neurologic Surgery, Mayo Clinic.
†Section on Ophthalmology, Mayo Clinic.

thru the meningeal connections with the venules. Certain observers believe that actual stomas exist between the veins and the villi of the arachnoid, while others believe that the endothelial wall of the arachnoid acts as a dialyzing membrane and permits the cerebrospinal fluid to escape into the venous system.

The phenomenon of choked disc caused by increased intracranial pressure is generally considered as the result of mechanical obstruction from intracranial neoplasm, inflammation or vascular lesions. Clinical observations demonstrate that lesions, such as brain tumors, which obstruct, either partially or totally, the normal flow of cerebrospinal fluid, produce increased intracranial pressure and unilateral or bilateral hydrocephalus with associated choked discs. Lesions which interfere with the normal absorption of the fluid, even tho the foramina and the aqueduct remain open, produce increased intracranial pressure and "communicating" hydrocephalus with associated choked discs. Other lesions, such as parasagittal tumors and posterior cortical tumors of the frontal lobe, produce increased intracranial pressure without hydrocephalus or choked discs. Tumors near to or at the chiasm are commonly associated with pale or atrophic discs, but rarely, if ever, with choked discs, altho they cause increased intracranial pressure.

The experimental work of Parker tends to prove the theory of mechanical obstruction, but Behr stresses the damming back of the cranially directed lymph flow thru the optic nerves, as the factor producing choked disc. Even tho one accepts the best portions of the numerous theories advanced concerning the cause of choked discs, there are many clinical problems encountered daily which remain unanswered. For instance: Why does choking of the discs develop more rapidly and to a greater degree with tumors obstructing the aqueduct of Sylvius or of the fourth ventricle than with supratentorial tumors producing equally increased intracranial pressure? Why do pale or atrophic discs without choking occur with chiasmal tumors when intracranial pressure is increased? Why are cortical tumors of the frontal lobe rarely if ever associated with choked discs, while anterior cortical and subcortical tumors of the frontal lobe commonly are? Why does high choking of the discs accompanying tumor of the posterior fossa subside more rapidly following surgical decompression

than low choking of the discs resulting from supratentorial tumor? If choking of the disc is due to mechanical obstruction, as shown by Parker, why does it not subside in two or three days after intracranial pressure falls to normal? Why do choked discs occur with localized leptomeningitis resulting from otitic and mastoid infection, when none or few of the general signs of increased intracranial pressure are present?

Therefore, inasmuch as ophthalmoscopic phenomena and neurologic symptoms occur as the result of increased intracranial pressure and hydrocephalus, independent of, or with the local symptoms produced by the neoplasm, we have attempted to record, by means of the apparatus referred to, the exact intracranial pressure with the corresponding clinical phenomena. A brain

Fig. 1. Burrs used for opening skull and die used for cutting threads on the margins of the trephine opening.

cannula is inserted into the lateral ventricle and is fixed in place by passing it thru a bone washer, which is screwed into a trephine opening in order to eliminate the factors of atmospheric pressure, and to prevent leakage of spinal fluid either about the bone washer or the cannula passing thru it. The cannula is then connected by a thick walled rubber tube to a glass manometer of sufficient length to record, in actual millimeters of cerebrospinal fluid, the exact pressure within the ventricle.

TECHNIC (ADSON).

The patient is placed on the operating table in the dorsal position, with the head rotated to the side. The area chosen for the trephine opening is approximately 4 cm. above the external occipital protuberance, and 4 cm. to one side of the

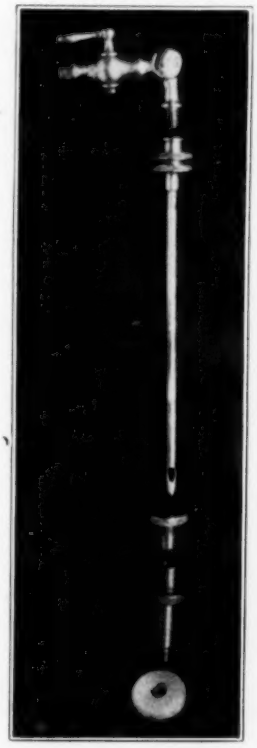

Fig. 2. Brain cannula, bone washer locking device, and elbow employed to fix the cannula at the desired depth within the ventricle, preventing movement of the cannula and leakage of cerebrospinal fluid.

median line. The skin is anesthetized as in preparation for unilateral ventriculogram. An incision is made in the scalp, sufficient in length to expose an area for trephining with the usual Hudson-Duran burrs, giving an opening approximately 15 mm. in diameter (Fig. 1). A steel die is then employed to cut a thread in the wall of the skull in the trephine opening, to prepare it to receive a beef bone washer which has had threads turned on its edge to fit those on the opening (Fig. 2). There is an opening in the center of the washer, of a size to permit the entrance of a brain cannula, 2 mm. in diameter. The center opening in the bone washer also has threads to admit a locking device, into which the brain cannula has been fitted before it is screwed in place. There is a thumb nut on this device, which makes it possible to lock the cannula in place at the depth desired, and prevents movement of the

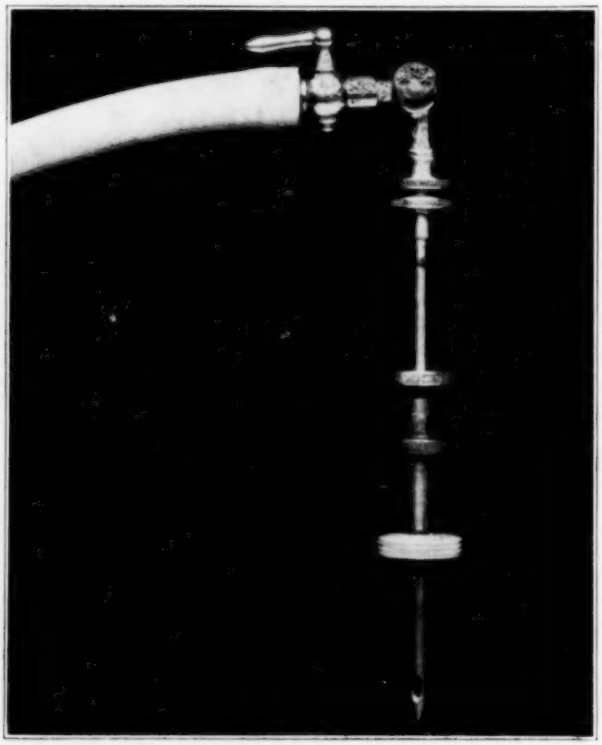

Fig. 3. Brain cannula with additional mechanical device which permits locking the brain cannula in place by screwing the bone washer into the trephine opening.

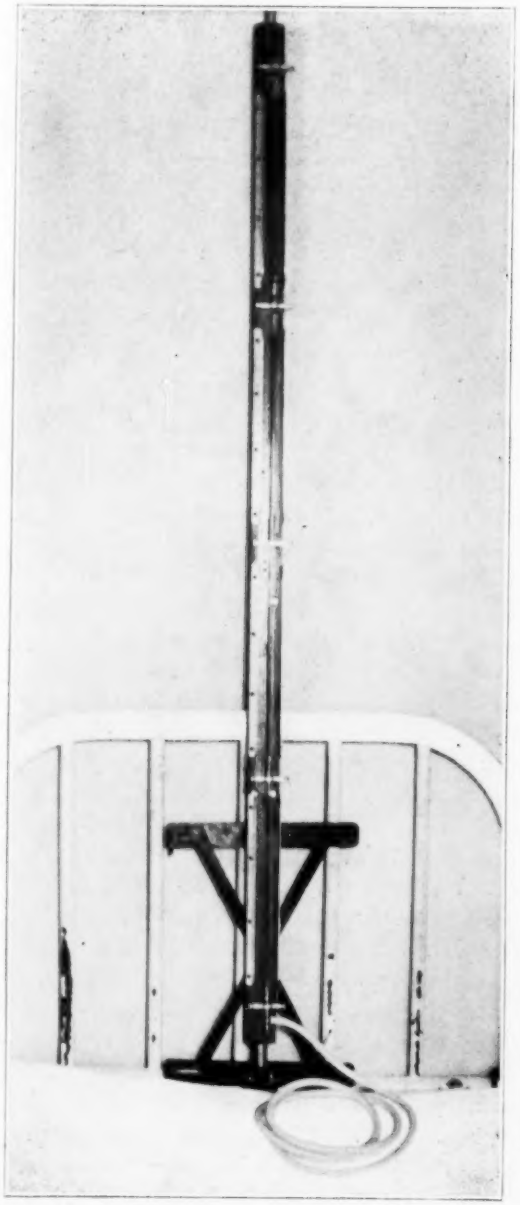

Fig. 4. Manometer with rubber connection and bed frame, permitting readjustment of position. Manometer records 1200 mm. of cerebrospinal fluid.

cannula or leakage of cerebrospinal fluid. The dura is then opened, and the brain cannula with its trocar is inserted into the posterior horn of the lateral ventricle; the trocar is removed, permitting flow of cerebrospinal fluid. When the increased pressure has been relieved, and the cerebrospinal fluid merely flows from the outer end of the cannula, the bone washer, with the locking device screwed in place, is slipped over the end of the cannula and adjusted in the trephine opening. The washer is then turned in until it is flush with the external surface of the skull and forms a rigid support. The thumb nut on the locking device is turned until the cannula is firmly locked within the bone washer at the depth desired. A small elbow, with a valve and a thumb nut, is screwed into the free end of the cannula to control the flow of cerebrospinal fluid (Fig. 3). The scalp is then closed over the bone washer. To remove the brain cannula, it is necessary only to unscrew the locking device within the bone washer, without opening the wound. The brain cannula and the locking device can then be removed readily, leaving a small opening between the edges of the scalp, which can be closed by bringing the edges together, under slight pressure. The wound is covered with a sterile dressing; the elbow, too, is covered temporarily with a sterile dressing. The patient is then returned to his room, and the cannula is connected to the manometer. The manometer consists of four calibrated 2.15 mm. pyrex tubes (used by Ayers in his spinal and cistern manometric readings) placed one on and into the other; they are fixed firmly to a frame, which is fastened to the head of the bed, and can be adjusted so that the lower or zero end may be placed at the same level as the intracranial end of the brain cannula. Thick walled rubber tubing, 2 mm. in diameter, is then connected with the manometer, after which a physiologic solution of sodium chlorid is injected into the free end until it is seen to rise in the manometer (Fig. 4). The free end of the rubber tube is then compressed to hold the solution in the tube and manometer, just preliminary to connecting the tube with the elbow attached to the brain cannnula. This procedure is carried out under strictly aseptic conditions. With the patient either in the reclining or in the upright position, according to the observation to be made, his head is adjusted to the desired level and the manometer is shifted to obtain the zero level (Fig. 5). The stop cock attached to the lower end of the manometer is

opened to permit the escape of excess solution. Ordinary precautions are necessary to prevent contamination of the brain cannula with its elbow. The valve in the elbow attached to the manometer is opened to permit cerebrospinal fluid to flow thru the tube into the manometer for recording. Examinations of the fundus and of intraocular tension are made as a routine before operation and during the course of the observations. Readings of the manometer are made every fifteen minutes, and blood pressure as well as pulse and respiration

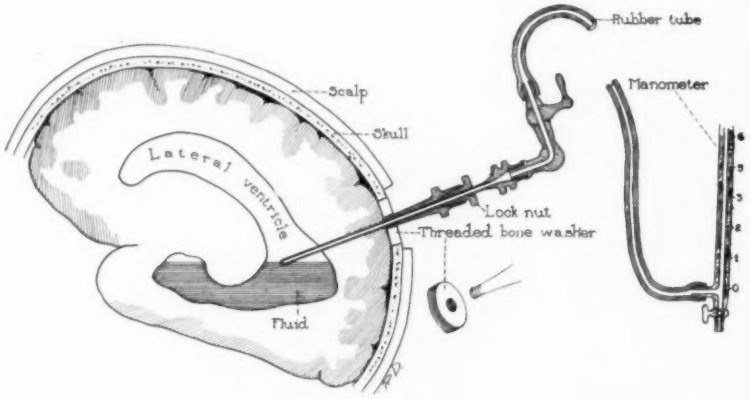

Fig. 5. Connections between the lateral ventricle, the brain cannula, the locking device, the rubber tube connections, and the manometer.

are recorded hourly. The physical states of the patient are carefully noted as they occur—falling asleep, awaking, straining, vomiting, eating, complaining of pain, having convulsions, becoming restless, or comatose, or the development of any other phenomena. Readings are made following the administration of various drugs. The readings on the manometer are checked from day to day with the numerous phenomena that are associated with the symptoms of intracranial pressure. Besides the daily readings, a record should be made of the ophthalmologic and neurologic phenomena when the stop cock is open at the bottom of the manometer, to permit escape of cerebrospinal fluid in the attempt to relieve the clinical symptoms.

Report of a Case.

A well nourished, well developed boy, aged sixteen, came to the Clinic June 27, 1927, complaining of headache, dizziness,

and occasional diplopia. The family history and the patient's previous history were negative. Six weeks before, he had first experienced frontal headache, which appeared on running or jumping, and persisted one or two hours. Two weeks later, the headache began to appear without previous provocatory exertion, lasted for twenty-four hours, and was associated with nausea and vomiting. The pain, as it occurred originally, was throbbing in character; recently it had become dull and aching, more or less continuous, and extended over the entire frontal area. Altho frequent periods of cessation occurred during the four weeks previous to his coming, he had approximately one severe paroxysmal attack each week, coming on in the afternoon, besides the more or less constant dull ache. Invariably, the paroxysmal attack was associated with vomiting. Recently, the headache had become rather pronounced over the right eye. The attack, from which he was suffering at the time of admission, had begun thirty hours before. Unsteadiness in gait on arising was noticed two weeks previously; it lasted only for a minute or two, but often he was compelled to catch hold of a piece of furniture to prevent himself from falling. Diplopia became manifest at the same time and was particularly noticeable when the patient looked directly ahead. He had had one attack of numbness in the fingers; he remembered the fingers of both hands were affected, those of the left hand more markedly. At the same time, there had been slight numbness at the corner of the mouth and along the margin of the tongue on the left side.

At examination, systolic blood pressure was 125, diastolic 90, and the pulse rate 66. The temperature was normal. The hemoglobin was 85 per cent, the erythrocytes numbered 4,960,000 and the leucocytes 9,400. The Wassermann test of the blood was negative. Urinalysis gave specific gravity 1.028 and an acid reaction. Roentgenologic examinations of the head and of the chest were negative. Vision was 6/7 on the right and 6/6 on the left, both with correction; the pupils were equal and the reflexes were normal. The fields were normal in form and the blue color field was normal, but the patient was blind to red and green, a trait which is familial and is present also in a brother. Fundus examination revealed bilateral, acutely choked discs of 3 diopters, with slight weakness of the right sixth cranial nerve and right homonymous diplopia. The intraocular tension July 9, 1927, measured by a Schiötz' tonometer, was 17 right and 17 left (Lillie).

Neurologic examination of the cranial nerves was negative except for diplopia and choked discs. The hearing was normal. Examination of the muscular and of the sensory systems was negative. Coordination was normal. There was a suggestion of bilateral increase in the patellar, achilles, and plantar reflexes, slight rigidity of the neck, and a slight suggestion of ataxia, with falling to the left when the patient attempted to sit on the edge of the bed. The type of gait could not be observed, because of the dizziness which manifested itself when the patient attempted to rise. Definite incoordination could not be demonstrated by the various finger-to-finger, and finger-to-nose tests. There was only a faint suggestion of a positive reaction to adiadokokinesis on the left.

On the basis of the history and observations at examination, we deduced that the lad was suffering from an intracranial neoplasm without definite signs of its situation. Dizziness and diplopia, with so rapid an onset, might suggest lesion of the posterior fossa; the single attack of indefinite numbness, with involvement of the sixth nerve on the right, might suggest a lesion of the right parietal lobe; the stiffness in the neck might be caused quite easily by increased intracranial pressure, or by lesion of the posterior fossa. The frontal headache, with exaggeration of pain over right eye, was not sufficient to be of value in locating the lesion. In the absence of definite cerebellar signs, we did not feel justified in advising exploration of the cerebellum, but hoped that we might be able to determine the site of the lesion by a ventriculogram and ventricular estimation. This was attempted July 11. The usual procedure was carried out. The posterior horn of the left lateral ventricle was entered and 20 c.c. of colorless cerebrospinal fluid was removed. The manometric reading of intracranial pressure before the fluid was removed was 120 mm.; it fluctuated, and dropped 28 mm. on a long inspiration and rose 15 or 20 mm. above 120 mm. on coughing or straining. During the removal of the cerebrospinal fluid, an equal amount of air was injected by the fractional method, that is, 5 c.c. of fluid was removed at a time and replaced by an equal volume of air. Subsequently, a general convulsion developed, the patient became unconscious, and it was necessary to remove the air immediately. Later, he regained consciousness, but remained confused; therefore we carried the procedure

no further. The brain cannula was held in place during the ventricular study. In the tabulation, the intracranial pressure as measured by the manometer is recorded, with simultaneous estimations of choking of the disc, intraocular tension, blood pressure, pulse, and respiration. The patient's condition became gradually worse, and death occurred July 15. At necropsy, an extensive glioma was found in the right parietal lobe.

Comment.

It is impossible to draw specific conclusions from so meager a study of intracranial pressure with ventricular estimations. In this case, hourly studies of the pressure are presented with the associated phenomena. It is interesting to note how decidedly the intracranial pressure varied in short periods without direct relation to the blood pressure, except that there were cycles of lowered systolic blood pressure, during which the patient was quiet or sleeping, occurring before a rise in intracranial pressure and pulse pressure, attended with slow respiration and pulse. The intravenous administration of glucose and the oral administration of magnesium sulphate apparently lowered intracranial pressure, for short intervals, while pituitrin and epinephrin caused a temporary rise in both blood pressure and intracranial pressure. Following the administration of codein, a slight drop in blood pressure was observed, but it is difficult to state whether this was due to the drug or to the patient's being asleep. Simultaneously with an increase in intracranial pressure, the patient would become restless, confused, and more or less irrational, and muscular twitchings with convulsions would occur. Drainage of cerebrospinal fluid thru the manometer failed to alter the neurologic phenomena, which could be explained, quite readily, at necropsy, by the absence of any appreciable degree of hydrocephalus. The manometric readings roughly indicated the actual pressure within the brain substance from time to time. The extensive glioma in the right parietal lobe found at necropsy apparently had blocked the aqueduct and had produced pressure on the crus cerebri at the incisura tentorii cerebelli, with prolapse of the medulla and of the cerebellar lobes thru the foramen magnum.

The intraocular tension remained unchanged, while the discs showed evidence of increased choking, with additional retinal hemorrhage. However, choking did not increase to

the maximum of 7 to 8 diopters, even tho the intracranial pressure reached 900 mm. of cerebrospinal fluid. At the time of respiratory failure, the intracranial pressure was recorded at 550 mm. (not its highest point). Death was due, no doubt, to indirect pressure on the respiratory center.

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MANOMETRIC READINGS OF INTRACRANIAL PRESSURE AND NEUROLOGIC AND OPHTHALMOLOGIC PHENOMENA.

Time	Pulse	Respiration	Blood pressure		Intracranial pressure	Remarks
			Systolic	Diastolic	Cerebrospinal fluid, mm.	
7-11-27 p. m.						
3:20	66		125	90		Ventriculogram started; local anesthesia.
3:40	140		105	75		Convulsion followed injection of air
3:45	148		95	65		Air withdrawn; cannula and wound closed; convulsions subsided; patient confused.
4:45	88	24	120	90	320	Attachment of brain cannula with manometer; headache relieved.
5:45	80	20	124	88	400	Restless; headache; *codein gr. ½.*
6:45	56	20	132	84	420	Quiet; severe headache.
7:45	58	18	138	79	300	Quiet; headache less severe.
8:45	64	16	130	70	250	Quiet and sleeping.
9:00					450	Awakened suddenly.
9:45	68	16	132	80	420	Awake and quiet; headache less severe.
10:45	80	16	120	68	280	Sleeping; manometer reading dropped to 120 mm. while vomiting at 10:00 p. m.
11:45	84	14	120	70	210	Awake and restless.
7-12-27 a. m.						
12:45	66	16	120	70	460	*Codein gr. ½* at 12:15 a. m.; fell asleep at 1:00 a. m.
1:15					600	Asleep; manometer readings fluctuating 450-600 mm.; pulse irregular.
1:45	70	16	124	48	400	Asleep; manometer readings fluctuating.
2:45	68	14	128	68	320	Asleep.
3:45	70	14	126	58	400	Quiet.
4:45	74	16	124	62	460	Quiet.
5:45	72	16	118	66	450	Quiet; manometer readings fluctuating.
6:45	66	16	115	60	280	Restless; *codein gr. ½* at 7 a. m.
7:45	60	18	122	70	520	Quiet; headache less severe; dozing occasionally.
8:45	60	16	110	70	290	Quiet.
9:45	72	20	114	68	225	Awake; trying to void urine; manometer readings fluctuating.
10:45	68	16	120	66	185	*Fundus examination*: choked discs 3 diopters both eyes; ocular tension 17 mm. in both eyes.
11:45	56	18	126	78	690	Restless; headache severe; manometer readings fluctuating 600-900 mm.
p. m. 12:45	52	16	136	80	880–980	Quiet; *codein gr. ½* at 12 m.; conscious; answers questions; cerebrospinal fluid blood tinged.

MANOMETRIC READINGS OF INTRACRANIAL PRESSURE AND NEUROLOGIC AND OPHTHALMOLOGIC PHENOMENA—(Continued).

Time	Pulse	Respiration	Blood pressure		Intracranial pressure	Remarks
			Systolic	Diastolic	Cerebro-Spinal fluid, mm.	
1:45	60	16	130	70	135	Vomited; manometer reading dropped after vomiting.
2:45	76	20	124	86	145	Quiet.
3:15					400	Wound dressed; pressure rose during movements of body.
4:00	58	16	110	88	138	Quiet and dozing.
5:00	68	18	120	88	135	Restless and disoriented.
6:00	64	16	132	78	145	Quiet.
7:00	76	20	124	76	130	Somnolent; *codein gr. ½.*
8:00	76	18	114	60	138	Quiet.
8:55					263	Administration of 100 c. c. of 20 per cent glucose.
9:00	68	16	120	50	263	Fell asleep and slept until 10:45 p. m.
10:55	96	16	108	72	178	At 10:45 p. m. manometer reading dropped to 178.
11:15						Restless; manometer reading fluctuating (200-520 mm.)
12 m.	92	16	106	70	320–646	Restless.
7-13-27 a. m.						
1:00	80	16	115	64	320	Sleeping.
2:00	76	16	112	66	364	Sleeping.
3:00	66	20	120	68	310	Restless; headache.
4:00	60	16	122	60	480	Restless; occasional hiccough; vomited.
5:00	66	18	120	62	320–642	Restless; *codein gr. ½* at 4:30 a. m.
6.00	70	16	124	78	400–580	Quiet; sleeping.
7:00	60	20	120	66	400	Restless; occasional hiccough; slept at intervals.
8:00	68	18	120	70	240	Sleeping.
9:00	70	16	110	70	180	Quiet and sleeping.
10:00	60	18	120	78	460–510	Quiet and sleeping; manometer reading fluctuating.
11:00	60	16	110	80	480–600	Manometer reading fluctuating synchronous with heartbeat, rising and falling 20-30 mm.
12 m.	72	16	114	88	500–600	Conscious and comfortable.
p. m.						
1:00	68	18	112	90	500–600	Sleeping.
2:00	72	16	120	80	165	Complaining of headache; *codein gr. ½.*
3:00	64	18	120	100	265	Sleeping.
4:00	76	18	100	90	290	Sleeping.
5:00	76	16	110	80	300	Awake and comfortable.

MANOMETRIC READINGS OF INTRACRANIAL PRESSURE AND NEUROLOGIC AND OPHTHALMOLOGIC PHENOMENA—(Continued).

Time	Pulse	Respiration	Blood pressure		Intracranial pressure	Remarks
			Systolic	Diastolic	Cerebrospinal fluid, mm.	
6:00	80	14	120	90	400–450	Awake.
7:00	80	14	116	90	285	Resting quietly.
8:00	80	16	118	78	360	Resting quietly.
9:00	96	18	118	72	600	Restless; tossing about in bed.
10:00	88	16	114	78	180	Sleeping but moaning.
11:00	110	16	115	70	210	Restless.
12 m.	68	16	120	78	460	Restless; *codein gr. ½* at 12:15 a. m.
7-14-27 a. m.						
1:00	60	16	124	72	520	Restless; headache and vomiting.
2:00	78	18	124	82	500	Quiet; short intervals of sleep.
3:00	90	14	120	74	250	Sleeping.
4:00	100	16	118	80	300	No response when spoken to.
5:00	88	16	120	90	340–400	Condition unchanged.
6:00	72	18	124	80	290	Manometer reading rose to 450 when patient was bathed.
7:00	80	18	128	86	450	Restless; evidence of pain; no response when spoken to.
8:00	68	14	110	86	345	Evidence of pain; hand kept on right side of head.
9:00	52	16	120	80	300–600	Restless; *codein gr. ½* at 8:30 a. m.
10:00	64	14	120	88	360–700	Restless; fundus reading 3-4 diopters; recent hemorrhage; intraocular tension 17 mm. both eyes.
11:00	60	14	136	90	390–450	Complained of pain with rise in manometer reading.
12 m.	64	14	130	80	440–500	Restless.
p. m. 1:00	60	14	130	80	400	Magnesium sulphate, 1 oz., by mouth.
2:00	84	16	134	90	303	Quiet; momentary convulsion at 2:10 p. m.
3:00	60	18	130	92	250	Quiet.
4:00	60	16	120	96	280	Quiet; momentary convulsion at 3:50 p. m.
5:00	72	16	128	80	310–320	Quiet.
6:00	76	18	138	94	440	Quiet.
7:00	64	16	114	84	400	Slightly restless.
8:00	68	14	128	92	420	Quiet.
9:00	92	18	124	70	200	Sleeping; **pituitrin 1 c.c.** intramuscularly at 8:38 p. m.
7-14-27 p. m.						
10:00	80	12	115	80	200	Sleeping; tips of fingers cyanotic.
11:00	98	12	120	88	380	Muscular twitching both arms.
12 m.	110		124	84	400	Restless; pulse irregular.

MANOMETRIC READINGS OF INTRACRANIAL PRESSURE AND NEUROLOGIC AND OPHTHALMOLOGIC PHENOMENA—(Continued).

Time	Pulse	Respiration	Blood pressure		Intracranial pressure	Remarks
			Systolic	Diastolic	Cerebrospinal fluid, mm.	
7-15-27 a. m.						
12:15	116	10	128	80	480	Restless; hiccough.
1:00	98	12	128	78	350	Quiet and sleeping.
2:00	108	14	124	76	332	Restless; evidence of pain; *codein gr.* ½ at 2:15 a. m.
3:00	66	12	130	70	432	Quiet and sleeping.
4:00	76	12	124	82	405	Sleeping.
5:00	68	16	120	74	400	Momentary convulsion.
7-15-27 a. m.						
6:00	80	14	130	88	370	Drowsy; no response when spoken to.
7:00	68	18	138	88	385	Restless.
8:00	60	16	130	94	380	Quiet.
9:00	100	20	160	110	380	Quiet.
9:45	140		116	100	550	Respiration ceased; artificial respiration begun and continued until 12 noon.
10:10	150		116	100	200	Pressure relieved by draining fluid.
10:15	150		116	100	235	*Atropin gr.* 1/100.
10:20	160		116	100	110	Artificial respiration.
10:30	172				5	Sudden drop in manometer reading.
10:30 until 12 m.	160–170				0	Kept alive by artificial respiration. Death with respiratory and subsequent cardiac failure at 12:00 noon.

DISCUSSION.

DR. WALTER R. PARKER, Detroit, Mich.: Dr. Adson has kindly referred to the experimental work I did some time ago, wherein the relation of the tension of the eyeball to choked disc was considered. This permits me to make a few remarks in regard to the work that was done at that time.

Our experimentation was conducted entirely to determine whether the relation of the tension of the eyeball to the first appearance of choked disc could be established. It was quite by accident that the possibility occurred to me, and it was after this fashion.

When the tenometer first came out, I was fortunate enough to get one quite early. In an effort to determine the average normal ocular tension, all the patients, staff, nurses and everybody who would submit to it, were subjected to a test by the Schiötz tonometer. One and a half years later, while reviewing a number of brain tumor cases, I discovered there were six in which the tension had been taken in both eyes, and I observed that in five of the six the swelling of the nerve was higher in the eye showing the lesser tension. It then occurred to me that the

mechanical theory of choked disc might be verified, and that the tension of the eyeball was a factor in determining which nerve would be first affected. Our experiments were conducted by trephining one eye in dogs and monkeys, reducing the tension appreciably. Intracranial pressure was then induced by artificial means. Time will not permit a recital of details, but we found the best results from using a brass disc, fitting it into the skull after the trephine opening had been made and the sponge introduced. It is essential that there be no leakage around the disc, as otherwise the intracranial pressure may not be sufficiently increased to produce choked disc.

In nine dogs and three monkeys, the experiment went on to a satisfactory conclusion, and in each of these cases, we were able to induce choked disc first in the eye that showed the lesser tension. Clinically this is not always easy to observe. Choked disc is an incident in the life history of brain tumor. The swelling gradually recedes and atrophy takes place. If a patient comes under observation while the swelling present in the first eye is receding, the nerve in the second eye may show a higher degree of swelling than the one in the eye with the lesser tension. It is therefore necessary to differentiate clinically between an early or late edema of the nervehead.

Dr. Suker made a statement with which I do not entirely agree. He said, that in all cases of acute hydrocephalus, there must be present at some time a papilledema, but my experience has not shown this to be true. I hope Dr. Adson in his closing remarks will give us the result of his observations in this regard.

The work that Dr. Adson and his associates have undertaken is colossal, and they are going to the bottom of some very perplexing problems. I think we are to be congratulated on having this preliminary report presented before the Academy, and we shall all look forward with great anticipation to the ultimate findings.

Dr. A. W. Adson, Rochester, Minn., (closing): I wish to express my sincere thanks to Doctor Parker for his generous discussion of my paper. Also, to emphasize that this investigation is carried out judiciously, with the consent of the patient, and only in such patients who present a complicated syndrome, and in whom it is difficult to make a diagnosis. It is our hope that, as we proceed with the investigation, we may learn something of diagnostic and therapeutic value in the study of intracranial pressure with its relationship to choked disc, intraocular tension, optic atrophy, and neurologic phenomena.

A CASE OF SYMPATHETIC OPHTHALMIA OBSERVED OVER A PERIOD OF SIX YEARS.

S. Hanford McKee, B.A., M.D.

MONTREAL, CANADA.

It is not my intention, in this report, to take up or discuss the merits of the different methods of treating sympathetic ophthalmia, especially the later ones, such as the use of diphtheria antitoxin or the Coro method, but simply to give the details of a very severe case of sympathetic disease, that finally reacted completely to the use of salicylate of soda internally and 3% atropin locally.

According to Gradle, quoting Schirmer, the first available reference to the fact that internal disease of one eye may depend upon the other, was made by Bartisch in 1582, who says, speaking of injuries to the eye, that these may be followed by shrinking of the eyeball, which is very painful, "and in this case the other eye is in great danger". In the first third of the 19th century, several writers, Demours, Wardrop, Lawrence, showed definitely that they recognized the existence of sympathetic ophthalmia as a clinical entity, and in 1818 Demours reported three cases, in which he definitely established the existence of sympathetic disease. In 1840, Mackenzie, in the third edition of his famous Treatise on the Diseases of the Eye, pp. 523-534, first named the disease and gave the first formal description of it. "Whenever I see sympathetic ophthalmia, even in the first stage, I know that I have to contend with an affection, which, however slight its present symptoms may be, is one of the most dangerous inflammations to which the organ of vision is exposed". Mackenzie's contribution was epoch making, and taken with the reports of Lawrence, Crompton, Prichard and Critchett, established a firm knowledge of the disease. In 1854 and 1863, Prichard and Critchett respectively urged enucleation as a curative and preventative of sympathetic ophthalmia. In 1863, Critchett said of this disease, "That which especially

claims interest in these cases is the tediousness, the insidiousness, and the obstinacy of the inflammation, the destructive influence which it exercises on vision, and the resistance which it shows to all kinds of treatment."

For many years, then, sympathetic ophthalmitis, sympathetic ophthalmia, sympathetic uveitis, a serous or plastic inflammation of the uveal tract of one eye due to effects of a similar inflammation in the other, has been well recognized as a definite clinical entity.

In nearly all cases, it is due to a perforating injury involving the ciliary region. In genuine cases, the first symptom, as pointed out by Mackenzie, is dimness of sight. This comes on insidiously, without pain or premonitory symptoms. Within twenty-four hours, there is generally found a slight circumcorneal congestion, with a few deposits on Descemet's membrane and very mild iritic adhesions. Without proper treatment, the inflammatory process quickly increases, with complete occlusion and seclusion of the pupil.

J. D., an adult male of 47 years, was injured by a small piece of steel entering the right globe on October 20th, 1920. Two or three days later, the foreign body was removed by magnet, and a small piece of prolapsed iris excised. The eye did not quiet to my satisfaction, but as the patient kept bothering about getting home, he was allowed to leave the hospital, to attend the out-patient department. He came to the out-door only fairly regularly. On November 22nd, that is less than a month after the injury, he was advised that he must again enter the hospital. He did not appear again until the 29th of November, when he was sent into the wards. At this date, the injured eye had all the earmarks of a plastic iridocyclitis. There was diminution of vision, marked photophobia and pain, severe tenderness in the ciliary region, and a tension of 18, Schiötz. Examination also showed the uveal tract of the uninjured eye to be already involved. In spite of the inflammation in the uninjured eye, I decided to enucleate the injured one (as I had followed that course overseas with satisfaction). Consequently, on the 30th of November, the day following his appearance at the out-door, he was sent into hospital and the injured eye enucleated. In this eye, which caused sympathetic ophthalmia, we have a pathologic lesion definitely in the uveal tract. The choroid is about twice the normal thickness due to massive infiltration. The vessels are

Fig. 1. Cross section of a normal eye, to show relationship in width between retina, choroid, and sclera.

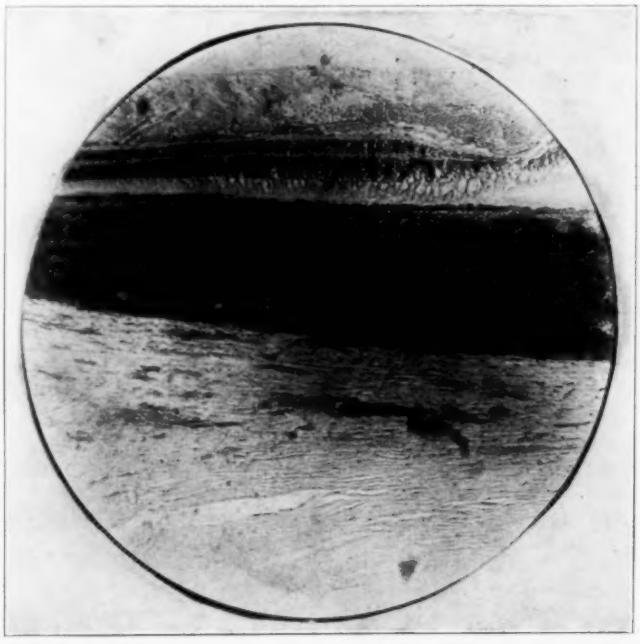

Fig. 2. Cross section of the eye which caused a sympathetic ophthalmia, showing massive infiltration of choroid.

dilated, and the sclera shows some inflammatory reaction about the blood vessels. The retina is edematous.

Following the operation, he was put upon the usual treatment of atropin 1%, hot compresses, diaphoresis, mercurial inunctions. In spite of this, the disease ran a most malignant course. The eye became most painful, iris bound down, and vision was reduced to hand movements. An attempt was made to control the inflammation by the use of neosalvarsan, altho his Wassermann was negative, but this treatment also had not the slightest effect upon the course of the disease. He was seen at odd times by Dr. Mathewson and myself, and finally we began instillations of 3% atropin, and put the patient on salicylate of soda, beginning with grs. xxx, three times daily. His mouth, which had been in a disgusting condition with pyorrhea, was also attended to. From this point the patient began to improve, the eye quieted, became free from pain, and his vision began to improve. Under this treatment, his eye completely cleared up, so that he was discharged from hospital, February 14th, 1921, cured. In May, 1924, the following note was made:

The patient has been seen by me at odd intervals since his discharge from hospital. He has not had any recurrence of inflammation, his vitreous is practically clear and his vision normal. Since that date the eye has remained perfectly well. I have examined him frequently. His eye now is normal. The vitreous is clear and his vision is 6/6, whole line.

References.

Gradle. American Encyclopedia of Ophthalmology, vol. XVI, p. 12369.
Critchett. Quoted by Randolph in Norris and Oliver, vol. III, p. 722.

DISCUSSION.

Dr. Chas. A. Bahn, New Orleans, La.: Especially to the younger ophthalmologist, Dr. McKee's contribution brings home, that one with great ophthalmic experience, both in civil and in military life, believes that the sympathetic ophthalmia patient has the greatest chance for recovery in the accepted methods of treatment—atropin, mercury, and the salicylates.

Most of us see very few cases. In reviewing our last twenty-five thousand office patients' records, I find that we have had but two with this disease; one, with favorable outcome, the other, not. In a serious and rare disease of this sort, it is so easy for the doctor and the patient to become panicky and lose the reasonable chance of future eyesight, by well meaning but misguided experimental methods.

Personally I do not believe that sympathetic ophthalmia is due to a single organism or cause, but rather to several entirely different causes which have a similar chemical action. A perforating injury is not necessary to the development of sympathetic ophthalmia, as illustrated in those patients with necrosing choroidal sarcoma, etc., who have developed the disease in the other eye.

Aside from the exciting cause, which is most commonly an injury, there are two other basic factors about which sufficient attention has not been paid. They are inherited and acquired sensitiveness to this particular disease.

I believe that a person might develop sympathetic ophthalmia (for example) from an injury received at the present time, who might not have developed this disease from the same injury received six months ago or a year in the future. The small number of cases reported during the World War was probably due not only to preventive enucleation, but also to the increased bodily resistance to this disease thru outdoor life, etc.

A study of the literature on sympathetic ophthalmia brings to mind two rather interesting observations. The first is, that the liability to the disease is very much greater in young persons. The second is, that the disease is apparently relatively infrequent in the negro.

Dr. Ben Witt Key, New York City, N. Y.: Dr. McKee's case is interesting to me from the standpoint of infection and pigment sensitization. If this is a case of sympathetic ophthalmia, which, without microscopic proof, is always challenged, then we have here another case to support the combined theory of infection and pigment absorption as the origin of sympathetic uveitis.

I presented a similar case last year before the Ophthalmic Section, New York Academy of Medicine, (published in the American Journal of Ophthalmology, August, 1926), which seemed to illustrate the all-important feature in the problem of the origin and clinical significance of sympathetic ophthalmia.

Briefly, the case was one of steel in the globe; the steel was extracted by magnet via the anterior route; the iris remained prolapsed in the wound at the limbus for several weeks; six consultations delayed removal of the offending eye, which finally became totally blind, the second eye being reduced to vision of 5/200. This, in spite of the usual eliminative treatment, foreign protein stimulation and massive doses of sodium salicylate, led to enucleation of the injured eye. Clinically, the case was one of sympathetic ophthalmia confirmed by several consultations. Subsequently, pansinusitis due to the pneumococcus capsulatus mucosa was discovered. Exenteration of the sinuses during three operative stages, each time demonstrated the effectiveness of the procedure by definite improvement in the sympathetic uveitis. One week later, mastoiditis developed, and mastoidectomy further demonstrated the toxic effect of the same microorganism upon the ocular disease. At the end of three weeks more, the eye was quiet and the vision 20/20, the fundus showing no visible destructive changes. Sections of the enucleated eye were pronounced those of sympathetic ophthalmia by Doctors Samuels, Verhoeff, and Finnoff.

This case, like Dr. McKee's, seems to confirm the theory of the combined action of infection plus susceptibility induced by pigment absorption. One may reasonably assume, therefore, that sympathetic ophthalmia

is a specific type of uveitis due to a focus of infection (or disease, of course including tuberculosis and syphilis), just as other types of uveitis have their origin. But the *clinical picture* and the *destructive* nature of the sympathetic type are due to the hypersensitivity and hypersusceptibility of both eyes from *pigment* absorption incident to uveal destructive changes from injury to the globe.

Dr F. Park Lewis, Buffalo, N. Y.: The history of the enucleation of the exciting eye in sympathetic ophthalmia is most interesting. It dates back to some time in the middle or latter part of the last century, when it was presumed that it would be helpful to enucleate the exciting eye, because in a condition which obtained in horses, called periodic ophthalmia, the veterinarians had found that if one eye became infected and that eye were removed, the other eye in their opinion did not become infected. It was assumed in consequence of that, that in sympathetic ophthalmia, if the exciting eye were removed, the other eye would be preserved. It happened, that last winter I had the good fortune to see a string of polo horses affected by periodic ophthalmia, which is an intense uveitis, and it occurred to me at the time, that if it were possible to discover the exciting organism of this disease, we might obtain a vaccine or a toxin which might be useful in uveitis in man. I took the matter up with Dr. Rosenow at Rochester, and he immediately fell in with the idea. We sent him a multitude of cultures from this string of horses—75. Twenty-three or twenty-four were affected; two were blind, and two had to be shot. I cannot go into the details and tell you how extensive and serious this disease is thruout the world. In some parts of Europe, one-third of the cavalry horses were lost. Dr. Rosenow was fortunate enough to find the exciting organism, discovering the microbe upon which it depended and the source of its origin, and made from it a toxin which he terms 701. He was good enough to send me some of that toxin in various strengths. It is exceedingly toxic. I saw it injected in moderate strength in a horse, and twenty minutes afterwards, he was so ill that he shook and wobbled and was unable to stand. We are now trying to determine exactly what the toxic dose is.

I have not seen any cases of sympathetic ophthalmia in recent years, but I have had several cases of chronic iritis which have received treatment, and I am glad to say that three of them have received definite benefit from the use of 701 in the proportion of 1 c.c. of the 1 to 100 strength.

This experience leads me to hope that we may find in 701 a specific for sympathetic ophthalmia, which so closely resembles that of moon blindness or periodic ophthalmia in horses.

Dr. Nelson M. Black, Milwaukee, Wis.: Dr. Bahn referred to the small number of cases of sympathetic ophthalmia that occurred during and following the World War. I have often wondered why more cases have not been seen and reported. May not the following possibly be the solution?

The men when they first went into service were vaccinated against smallpox; shortly thereafter, they were given an antityphoid injection; if they were wounded, they were immediately given an antitetanus injection. Is it not possible that as a result of the introduction of the considerable amount of foreign protein into the system, there was an immunity

produced against sympathetic ophthalmia? As a result of the above thought, we have used, in cases of penetrating wounds of the eyes, either milk injections, antityphoid, or antitoxin, as soon as possible following the injury, as a prophylactic measure.

Dr. Albert E. Bulson, Jr., Fort Wayne, Ind.: Sometimes there is a medicolegal side to the question of the enucleation of the exciting eye. Two of my ophthalmologic friends have been threatened with malpractice suits in cases where enucleation of the exciting eye was delayed and sympathetic ophthalmia developed.

I have taken care of six cases of sympathetic ophthalmia, seen first after development, in each of which the exciting eye was enucleated too late, and in every one of which unfavorable results were secured, notwithstanding the fact that heroic treatment with salicylates, iodides and mercury by inunction was given. Two of the patients were children who are now in the Indiana School for the Blind.

Recently, I saw a young man suffering with sympathetic ophthalmia, who apparently recovered as the result of the use of sodium salicylate intravenously in large doses. I would like to emphasize the value of such treatment. Another one seems to have made a complete recovery following blood injections used as a foreign proteid.

In my judgment, one rule should be followed in cases of perforating wounds in the ciliary region, and it is as follows: It is perfectly safe to watch the injured eye for a period of two or three weeks, possibly four, providing the injured eye apparently is going on to recovery. If the injured eye shows signs of continued irritation or inflammation, accompanied by tenderness, the patient is better off without that eye than with it, and you may save yourself a malpractice suit by urging enucleation of the exciting eye in such instances. I am sure that a majority of the cases of sympathetic ophthalmia that I have seen, have been due to delay in removing an exciting eye that showed no sign of improvement following prolonged treatment.

Dr. W. W. Pearson, Des Moines, Iowa: I did not intend to discuss this paper, but I feel I am justified in saying a few words and in reporting a couple of unusual cases.

One case was that of a railroad engineer, aged thirty-eight or forty. His water gauge blew up, with the result that he had a severe sclerocorneal wound, which healed in a comparatively short time after the excision of a portion of the iris. The resultant vision in this eye was about 20/24 with a cylindrical lens. He was dismissed with the warning that he might have sympathetic ophthalmia. He returned in about six weeks, with a depression of vision in the good eye and an inflammatory reaction in the injured eye. This was about sixteen years ago. The reaction in the injured eye was so acute and so severe, that we felt justified in removing it at once. The process in the sympathizing eye went on slowly until, after a few weeks, he was unable to tell time on an alarm clock. Fortunately, this gentleman and his wife were both of the type who followed instructions exactly. He was put to bed, was given a one hour sweat every day, and salicylates, varying from 60 grains three times a day to as high as 100 grains at a dose, were administered, the size of the dose being governed, in a measure, by tinnitus and the depression of

hearing. He continued this treatment for over a year, with the result that he had almost normal vision.

I watched him during the succeeding years, and his vision today is better than 20/24. He has been a useful member of society and has conducted his business successfully. One of my assistants discussed this case, following its recovery, with Dr. Verhoeff of Boston, with whom he was doing postgraduate work. Dr. Verhoeff said that, in his judgment, the patient's vision would fail. Such, however, has not been the case, and the period of observation has been about sixteen years. I do not regard that man's condition today as a fixed condition. I fear something will develop. He has a little photophobia, but at no time has he had an acute inflammatory flareup or sudden alteration in vision since his apparent recovery. Ophthalmoscopic examination reveals slight imperfections in the periphery of the lens, some floaters in the vitreous humor, and some evidence of old trouble in the uveal tract, in which the retina has participated. I have feared he may develop further changes in the lens.

Another case is one following cataract extraction in a woman about sixty years of age. She was a diabetic. She left the hospital in about ten days. There was no inclusion of the iris or capsule, and she returned home with no reaction justifying any suspicion of trouble. Within a couple of weeks, she returned with an inflammation of the operated eye, which was followed, in a few weeks, by an inflammation of the second eye. The inflammatory process in the operated eye drew the iris across the pupil and closed the iridectomy wound. Both eyes were involved, and since the eye in which I had done the cataract extraction several years before was quiet, I excised the central portion of the stretched iris, and she now has 20/50 vision. The unoperated eye developed such a painful condition, with loss of light perception, that it was removed about two years ago. The operated eye had been quiet over a period of years, so that I felt justified in making the new pupil.

I recognize that the classification of the second case as one of sympathetic ophthalmia is open to criticism. But the oculists who saw it so regarded it.

Dr. Robert von der Heydt, Chicago, Ill.: I am asked to state what I think of the slit lamp findings. I have had the opportunity to observe quite a few definitely clinically diagnosed cases of incipient sympathetic inflammation.

They do not differ in their incipient symptomatology from those of iridocyclitis due to other causes—this in spite of the pathologic anatomic differentiation of Fuchs, that is, the finding of lymphocyte foci in the choroid.

They differ from one another, some presenting a leucocytosis into the aqueous, others an increased viscosity and pigment phenomena.

Many years ago, while anticipating and watching for sympathetic inflammation, we were stimulated to closer observation. Among the results of this, were the findings that the near point receded, and that there were changes on the posterior cornea, which stained with fluorescein. I think these are also part of the symptomatology of other types of iridocyclitis.

The slit lamp has been a most wonderful aid in the diagnosis of the very incipience of iridocyclitis, irrespective of types and etiology.

Dr. S. R. Gifford, Omaha, Neb.: I would like to say a word of caution in regard to the use of intravenous injections of sodium salicylate. The caution is not as to the danger involved in its use, but a caution lest you fail to give the salicylate in large enough doses. If you give an intravenous injection, you can give only 20 to 30 grains. When you give oral doses, you can give 150 grains a day safely. The absorption may not be quite so rapid, but it is very rapid, as I was able in animals to obtain a test for salicyl in the aqueous twenty minutes after a dose had been given by stomach tube, and to estimate it quantitatively after 40 to 60 minutes. I have given intravenous injections in some cases where a rapid effect seemed especially desirable, but have at the same time given large doses by mouth, and I think the latter ought always to be done if you are depending on salicylate therapy.

Dr. Finnoff mentioned a case that had a late recurrence. We saw a case six years ago in which the eye quieted down under this therapy. He has had six or eight recurrences, the last within a year, and in each we have obtained the same good effect with salicylate therapy, the patient now having 20/30 vision. We have also looked for foci of infection in his case, and he has had several teeth and his tonsils removed. We have not seen the dramatic results from the removal of focal infections that are reported by some men, but look for them in cases which seem to hang fire. It is hard to figure out why there should be any relation between such foci and sympathetic ophthalmia, unless we believe a metastatic infection is the cause of the latter. If there should be a focus containing a virus or other organism that might cause sympathetic ophthalmia, and this organism lands in the eye that has been wounded, then it is conceivable that removal of the focus might influence the future of the second eye.

Dr. S. Hanford McKee, Montreal, Canada (closing): My object in bringing a case report of this kind before the society was in the hope that the matter would be discussed well by the members, and so brought to the minds of the members of the Academy, especially the younger ones. With the explosive engine everywhere as it is today, those of us who have not had the advantage of industrial work are meeting cases of very severe injury to the eye, and I think most oculists' practice in that respect has changed materially in the last few years; consequently, the problem of sympathetic ophthalmia, which seems to have been in the background for a certain period, I am afraid will be brought to our attention more than in the past.

I would like to say to Dr. Black, that I think he should add to the reasons why our soldiers have developed so little sympathetic ophthalmia, that they were well looked after by men competent to take care of injured eyes.

I wish to thank the Fellows very much for their discussion which this case report has brought out.

CONCERNING OBSERVATIONS OF THE SHARPNESS OF VISION OF ABNORMAL EYES WHEN TESTED AT A DISTANCE AND AT NEAR POINTS.

ALBERT C. SNELL, M.D.

ROCHESTER, NEW YORK.

The determination of loss of vision at a near point or at a working distance has become important since workman's compensation laws have become general. In view of the fact that tests for visual acuity are commonly made at a distance of twenty feet or six meters, whereas skilled workmen use their vision at a near point, usually between fourteen and twenty inches, the question has arisen whether or not the distance test alone is an accurate measure of the form sense and the corresponding visual efficiency. Therefore an investigation of the possible differences between the form sense at near and at a distance has a very practical application as well as a scientific interest. Since the acceptance of the report of the committee on compensation for eye injuries by the American Medical Association in May, 1925, our attention has been directed to the practical necessity of making near tests of visual acuity, and since this time we have given the subject careful study and have made observations on more than two thousand cases. This paper gives briefly a summary of our observations and deductions, and suggests some possible practical applications.

DIFFICULTIES ENCOUNTERED IN MAKING THE NEAR TESTS.

In order to make accurate tests at the near point, a standard scale must be employed uniformly. The standard scale for testing near acuity, or for determining reading ability, must conform to the Snellen formula in exactly the same manner as does the scale for measuring distance acuity.

We insert here the Snellen formula for expressing acuteness of the central form sense. This will serve to keep in mind the principles embodied in the Snellen scale, and will

clarify an important point which we shall make later on. Every correct scale of test letters for any distance and for all distances, whether near or far, must be made in conformity with this formula.

Snellen Formula: S (or V) $= \frac{d}{D}$ in which d equals the distance at which the letters are read, and D the distance at which the letters subtend an angle of five minutes, and the greatest distance at which the normal eye can read them. S = Sehschärfe and V = *acuity* of vision.

The American Ophthalmological Society suggested a slight modification of the formula for expressing near acuity, as follows:

$L = \frac{d}{D}$, in which L = lectio (reading power) as, more precisely, the formula for near expresses the relative accuracy of one's reading ability.

In order to find whether the near test cards in common use conformed to this scale, ten different near cards, published by eight different supply houses, were collected and studied. This study revealed a wide divergence in nomenclature, in kind and style of fonts, in distinctness of print impress, and in the size and character of the small lower case letters, when these were used. No one card seemed to conform to the standard in every respect.

In addition to the lack of a uniform standard scale, other difficulties were encountered, inherent in the nature of the near test, which made accurate tests of the near acuity most difficult. These difficulties are described clearly by Professor Snellen[1]. Professor Snellen states, that in determining visual acuity, "we measure the visual angle for distance in order to obviate the influence of the power of accommodation or the necessity for glasses for focusing near objects." He further states, that another advantage in distance testing is found in the fact, that the larger objects are more easily constructed; and besides, the exact distance at which the test is made is relatively less likely of error for distance than for near. He concludes, "Consequently, tests made at a distance are in all respects more accurate than those made at short distances." We shall proceed to discuss these difficulties in detail.

NOMENCLATURE: A survey of these collected near test cards reveals that there is in use no common nomenclature, notation or method of expressing visual acuity for near or reading abilty. This lack of a common language leads to confusion, misunderstanding and error. For example, we find the following notations on these cards to designate their progressive increasing size.

1st, Jaeger 1, 2, 3, 4, 5, 6, etc.;

2nd, .50 D, .75 D, 1.D, 1.25 D, 1.50 D, etc.;

3rd, + .50 D, + .75 D, + 1.D, etc.;

4th, D — .50, D — .75, D — 1., D — 1.25, etc.;

5th, .50 M., .75 M., 1. M., etc.;

6th, the simple numbers 4, 5, 6, 7, etc., indicating decimeters;

7th, the arithmetic series 1, 2, 3, 4, 5, 6, etc.;

8th, V = .50 D, V = .75 D, V = 1 D, V = 1.25 D, etc.

9th, 20/15 Sn., 20/20 Sn., 20/30 Sn., etc.;

10th, printers' nomenclature, such as 4 point, 5 point, 6 point, etc., and also such as diamond, pearl, etc.

Some of these designations obviously have common corresponding values, but many are purely arbitrary, and the size of the visual angle cannot be deduced until their visual angles are determined.

The Jaeger system of notation made use of the various fonts of printers' types which were in use in his day. Both the type and the nomenclature are now obsolete, and this system conveys no mathematic meaning relative to visual acuity or to reading ability.

The significance of the capital letter "D" which is used on many cards is commonly misunderstood. Note above the many different expressions. The "D" is writen with a decimal fraction before it or after it, with a + sign before, with a dash or a minus between the D and numeral, and with a notation signifying an equation (V = .50 D, etc.). What do these symbols mean? In the first place, let us find out the significance of the "D". There is a prevalent notion that this "D" is a symbol for diopter. Even those who are best informed have wrongly interpreted this symbol to signfy diopter. There may be some explanation for this misinterpretation, since Oliver states, when describing the "sequence" of "type" in his metric near test letters, that a *size of type* is reached "equivalent to two dioptrys." And further on he

writes of a separation of "one half dioptre" between the graduation of type.[2] Oliver makes the metric measure and the diopter measure synonymous. It is easily comprehended that this is a fallacy, when one considers the definition of *diopter,* which is:[3] "Diopter: The unit of refraction of lenses, being the refractive power of a lens of the focal distance of one meter." Thus it must be apparent that diopters refer entirely to *refractive focal power,* whereas the meter is simply a unit of measure.

Altho the unit of focal distance of the diopter is one meter, the dioptric focal distance is in inverse ratio to the metric measure. The smaller the diopter, the greater the focal distance. For example, a .50 diopter lens has its focal point at two meters, a 2 D lens has a focal point at .50 meter, etc. The opposite is true of the size of test letters—the larger the letter, the larger is its visual angle, and the greater is the distance that it can be seen. A .50 meter line of test letters is one half the size of a meter line, etc. Thus the size of type or sequence of graduation of different lines of type cannot be associated with diopters. Neither can the metric distances or sizes of the test letter refer to diopters of accommodation, since diopters of accommodation, like diopters of lenses, have focal power in inverse ratio to their numerical expression. (See Prince's rule.)

As is well known, "D" in the Snellen formula signifies the greatest distance at which the normal standard eye can just distinguish the respective letters or characters as they increase in size on the card. Since the Snellen standard for normal acuity is a five minute character, the numerals adjacent to the "D" express the distance at which these letters subtend a five minute angle. These distances are commonly expressed in centimeters or in decimeters on the near cards, and the "D" on the near card has exactly the same significance for near as for distance, and is the "D" in the Snellen formula:

$$S = \frac{d}{D}.$$

From the elucidation above, it must be apparent that a + or — sign before the "D," or its accompanying fraction, is meaningless and absurd. The expression V = .50 D, etc., would seem to mean that visual acuity is .5 normal. Of course, this interpretation is incorrect, since ability to read the .50 D letters at 50 centimeters expresses an acuity of 100%. .25 D

read at 25 centimeters does not indicate 25% acuity, but 100% acuity when read at 25 centimeters, etc. If this equation is intended to convey an expression of acuity, it is necessary to assume or to supply the numerator of the Snellen formula, that is, the distance at which this size letter is read. Then only has the expression any mathematic significance.

In regard to the use of other symbols, such as "M" for meter, or the names of printers' fonts, we wish to remark that the use of "M" to express meter is sound and is not easily confused with diopter, but the use of modern names of the printers' fonts is not scientific, as these letters do not, as a rule, lend themselves to exact measurements of the visual angle, and do not convey any definite idea as to their relative size.

To prevent the possibility of confusion, the letter "D" should not be used in the notation of near cards, even when its meaning is understood. Its use should be confined to the refractive power of lenses and to the power of accommodation. Near cards are most convenient and accurate when numbered and printed so as to conform to the decimeter scale as recommended by the American Ophthalmological Society.

Fonts: In all of these cards examined, the small case fonts were not constructed with equal thickness of limbal parts or with equal open spacing between the limbs, and thus do not conform to the Snellen standard. The impossibility of selection of letters, the great dissimilarity of the small case letters as they appear in the printed words, renders such conformity impossible. In the main, small case letters, as found in ordinary print and as reproduced in these near test cards, have been constructed largely for their legibility. Thus they do not lend themselves easily to concise measurements of the visual angle or for the determination of visual acuity, but are best suited for testing reading ability, whereas near test cards having capital letters, similar to the distance cards, arranged individually or in words, afford a more accurate means of testing acuity. These, of course, should be constructed on the principle of five minute characters having one minute limbs.

In addition to these intentional and inherent irregularities in construction of fonts, the print impress of the smaller letters often causes considerable alteration in the various strokes, angles, and openings of the letters. This is another source of error.

Measurements of the Visual Angle for Near.

In order to determine the probable visual angle error in these collected test cards, we have made measurements of the size of the various characters. The individual letters and limbal parts were measured with a low power microscope, using a micrometer scale. The scale was so divided that 1/10 of a millimeter was divided into twenty parts. Thus each division in the scale represented .05 of a millimeter. With this scale we measured the letters contained in the smaller lines of the test cards, ranging from .37 m. to 1 m. Extended and more careful observations and measurements were made of the letter "e" in the 50 cm. size of test letters or its equivalent on the other cards, for purposes of comparison. Our measurements of the letter "e" covered the letter as a whole, each limb of the letter, and the spaces between.

The following interesting facts were noted: Taking the letter "e" as a whole, we found a considerable degree of accuracy. The standard for this letter would require a letter .73 mm. high. Its actual measurements in all eight cards were as follows: One card 16%, three cards 9%, and three cards 2% oversize, and one card 2% undersize. Thus four out of eight cards may be said to have conformed to the standard, a 2% variation being most difficult to measure.

The measurements of the limbs of the letters "e" showed a much greater variation from standard than did the size of this letter. Each element of the limbs and each space theoretically should measure .146 mm. On none of the eight cards did we find the printed "e"s uniform in their limbal parts. In three cards, the thickness of the limb varied from .1 mm. to .4 mm.; in three other cards, from .2 mm. to .4 mm.; in two, .15 mm. to .3 mm.; and in one, .2 mm. to .3 mm. In one card, the largest part was 175% too large as compared with the standard. In every "e" measured, some part of a limb was at least 100% too large.

Even a greater error was found in the open part of the "e", especially the lower open element. The respective measurements of the upper open part were: for four cards, .4 mm.; for two cards, .3 mm.; for one card, .5 mm. The measurements for the lower openings were: for two cards, .8 mm.; for one, .7 mm.; for two, .6 mm.; for one, .5 mm.; and for two, .4 mm. The latter, the smallest lower opening, was thus 175%

larger than the theoretic standard measurement of .146 mm.

When these measurements were made of the letters smaller than the 50 cm. ones, a much greater error was found. In many of these characters, an upper open part of the "e" was completely obliterated. This, of course, caused an absolute 100% error in the letter. This error occurred most often in the letters "a", "e", and "s". In the other letters, an error of 50% was often observed. When one realizes that a .37 m. letter requires limbal parts of exactly .1 mm., it is easily understood how a very small error in print causes a large percentage deviation from standard; a .05 mm. error becomes a 50% fault. Conversely, in the larger letters, those of one meter and larger, the same size error of printing and construction becomes relatively and proportionately unimportant and insignificant.

These observed facts must clearly demonstrate, that if our measurements of visual acuity for near and its efficiency or mathematic value are to be determined therefrom, a uniform and comprehensible nomenclature, a standard scale, and a common method of expressing and recording must be employed. All of these requirements are met when acuity for near is measured, notated, expressed, and recorded in conformity with the Snellen formula. Near acuity should be determined and recorded exactly as is distance acuity.

In conformity with this dictum, I have found the following method, which we use routinely in our practice, to be simple, practical and scientific. It records all the observed facts of the near test, from which one may not only deduce the relative acuity and efficiency, but also the power and available region of accommodation.

RECORD FOR NEAR READING ABILITY,
ACCOMMODATION, AND ACUITY OF VISION.

$$nV^a = \frac{\overset{*}{\|}\ \overset{\dagger}{\|}\ \overset{\ddagger}{\|}}{\underset{\|}{25\text{-}32\text{-}50}} = \frac{d}{D}$$

d = distance in cm. at which the print is read.

D = distance in cm. at which the letters subtend a five minute angle.

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* = punctum proximum.
† = punctum optimum (best reading point)
‡ = punctum remotum.
Figures are in centimeters.

The simplified notation for the records is expressed thus: $nV^a = \frac{25, 32, 50}{50}$ with + 1.25 S. added B, or $\frac{25, 32—34, 50}{50}$ with gl., or $nV^a = \frac{30, 35, 40}{75}$ with correction; the denominator being always the distance in centimeters at which the visual angle subtends a five minute angle.

It may be observed, that in making our distance tests for acuity, the numerator does not change, since in practice our distance tests are made at a fixed distance—6 m. or 20 ft.—whereas the near tests of acuity are necessarily made at different distances—10 to 50 cm., more and less—so that in the latter testing, the numerator is always changing. Also it may be observed, that in recording distance acuity, the denominator is subject to change, depending on the size of letters read at the fixed distance, whereas the denominator recording near acuity does not change in any individual record; but any practical denominator may be chosen. The denominator designates the distance at which the characters subtend a five minute angle, and denotes the line of letters actually read. This is best expressed in centimeters, altho the inch scale may be used.

When the metric system is used in recording tests of distance acuity, the same system should be used for recording the near tests, and when the foot system is used for recording distant acuity, the same system should be used for recording the near tests. The reasons are obvious.

Clinical Observations.

Before proceeding with the study of abnormal cases, we made numerous observations on patients with normal acuity, in order to determine, first, whether the near acuity in such cases was identical with the distance test; and second, the influence of age and of accommodation. In making these clinical observations for near, we chose the near test card which most nearly conformed to the Snellen formula. All measurements were made under the same conditions of illumination, with the same card, and in each case the near point was very accurately measured with a rod divided into centimeters. The measuring rod was held 14 mm. in front of the cornea, as recommended by Duane. The distance acuity was found in

the usual way. For both distances, errors of refraction were corrected.

We shall not tabulate all of these observations, but shall record the more important conclusions deduced from them. When visual acuity was not better than 6/6 for distance, very often it was with great difficulty that such a patient was able to read at the near point a character having the same visual angle. This was especially true of patients over thirty-five years of age, and also of those under ten. These patients could correctly name individual letters and words, especially capitals, but often could not read the small case print (Oliver near card used). This observation clearly demonstrated that there is a difference in the test for best visual acuity and the ability of a person to read. Ability to read freely requires in the printed line a larger visual angle than is required to identify single letters or words. One's reading ability is indicated by the punctum optimum in our formula, and the best acuity is expressed by the punctum remotum of our formula. Thus, if one wishes to be accurate in determining the best visual acuity for near, it is necessary to confine the observation to single letters, block capitals being best for this purpose, and determine the remotest point at which they can be identified. The difference in ability to read and the actual visual acuity for near, tested in this way, varies as much as 50% in size of their respective visual angles.

Accommodation is another important factor which has a modifying influence on acuity for near. Nearly all the patients observed, without regard to age, increased their ability to read and their visual acuity for near when their accommodative power was actively employed. Since it is imperative to find the best acuity for near, just as for distance, the test must be made at the distance indicated on the card. When such card is given to patients and they are asked to read, almost without exception, because of this desire to employ accommodation and also to enlarge the visual angle, the card is brought much nearer to the eyes than is indicated on the card as the standard distance for the particular line selected. This, of course, will give an erroneous idea of acuity, the visual angle being often double at the reading distance. Neither the punctum proximum nor the punctum optimum give the best acuity. Near acuity of vision is determined by finding the greatest distance at which the smallest

readable type can be just distinguished. This is the punctum remotum of our formula.

When the above rule is strictly followed and the test for near acuity is made by employing individual letters, preferably block capitals, and due attention is made for the use of any remaining accommodation, the near acuity and the distance were found to be identical in normal eyes. It seems reasonable to assume, that when the near tests are made with eyes having subnormal vision, the observations may be regarded as accurate, provided that the difficulties which we have pointed out are met, and meticulous care is observed in conducting the tests. In making our observations on abnormal eyes, we have followed the principles enunciated thus far in this paper.

Observation of Abnormal Cases.

Our observations were made on all conditions of abnormal eyes, including myopia, conical cornea, amblyopia, vitreous opacities, glaucoma, cataracts, and corneal scars.

The myopic cases which we have carefully measured revealed an unsuspected relationship between the distance acuity and near. I had had the feeling that myopic patients could see better at near than at distance, and I find that this opinion is commonly held. Landolt[4] states, that he has found that in high myopia, the visual acuity for near may be double that obtainable for distance. He also points out that theoretically the near acuity may be calculated from the distance, but not so practically. Our observations would indicate that considerable modification of such a view must be made in order to conform with the facts observed. In approximately 60% of our myopic cases under fifty years of age and with myopia not greater than —6D, either with or without normal acuity for distance, the smallest distinguishable character for near was larger, comparing visual angles, than was the smallest distinguishable character at six meters. This means, of course, that visual acuity was relatively poorer for near than for distance. This observation applies to myopic patients with the use of their glasses for each test, and for those having accommodation. Forty per cent of these cases had identical acuity for near and for distance. Where presbyopia necessitated the use of the addition of plus lenses, the situation was found to be mixed. Some of these cases had

slightly better acuity for near than for distance. In those myopic patients where myopia was greater than 8 D, acuity for distance usually being below a normal standard, there was *apparently* a better acuity for near than for distance, but invariably the near point was of necessity within a 25 centimeter range, and glasses of less focal power had to be used than the distant correction. Of course, when the near test was made with glasses of less dioptric strength than those used for the measurement of distance acuity, the size of the visual angle was larger for this reason alone. But even in this class of myopic cases, the actual differences in the size of the visual angle was slight. The difference in visual angle was usually 2½ to 5 minutes. The very near approach of the object to the eye enlarges the visual angle at a rapid rate, so that these measurements are most difficult in the high myopias. There were several cases of high complicated myopia in which the near acuity was considerably better than the distant, the visual angle being double.

The usual understanding that myopic patients see better for near is, of course, true as a practical observation, but it does not hold generally when the test relates to the comparative size of the visual angles where identical myopic correction is worn.

Conical Cornea: Eighty case records of conical cornea have been analyzed. This list includes all of my private cases. Of these, 20% had better near acuity than distant, and 80% had the same acuity. In only one case was there recorded a better distant acuity than near. These observations were made in every instance with the use of the best correcting glasses. It should be stated, that in a number of these cases which had a marked defect in acuity both for distance and for near, the glasses worn had the near focal point very near to the eye, thus increasing the visual angle as in myopia. We found that the sixty-three cases in which no difference of acuity was observed are, as a rule, classed among the less severe types of keratoconus.

Amblyopia: No material difference between the near and distant acuity has been observed in the study of amblyopic cases, when the acuity of each is determined by comparing the size of their visual angle. However, many of these cases, especially among the younger patients, apparently had better acuity for near than for distance. This discrepancy is due

again to the fact that these patients have often formed the habit of holding near objects very near to their eye, thus producing a large visual angle and enabling them to see correspondingly better.

VITREOUS OPACITY: No actual difference between near and distant acuity was observed in this class of visual defect. I have seen only one case in which the near acuity was less than the distance, assuming again, of course, that the comparison was made with the determined visual angles.

GLAUCOMA: These cases were not carefully tabulated, but we note in very many either a great difficulty in sustained reading, or that it was impossible. Apparently in many cases, visual acuity for distance was better. However, most of our observations were made while inflammatory processes were still active, which apparently accounts for these observations, and we had not the opportunity to separate the noninflammatory, quiescent cases from the active.

CATARACTS: Observations have been made of the relative difference of near and distance acuity in all cataracts seen by us during the past two years, but only a recent series of twenty-five consecutive cases is used for the following percentage determinations, as it was only in these latter cases that our observations were made with identical method of testing. Eleven of these were nuclear cataracts. Acuity for distance in these cases ranged between 6/9 and 6/60. Eight of the eleven, or 83%, had positively better acuity for distance than for near, and two, or 18%, had the same acuity, and one, or 9%, had better acuity for near than for distance. Fourteen out of the twenty-five cataracts were classified as zonular, coronal, peripheral, and as having diffused opacities. Eleven of these latter had better distance acuity, and three had either slightly better near acuity, or no difference was found.

In all of these cataracts where we were able to demonstrate a difference between near and distance acuity, the size of the pupil played an important part. When distance acuity was the better, the contraction of the pupil associated with accommodation was the cause of the poorer near acuity. This was demonstrated by the fact, that in all of these cases, the distance acuity likewise decreased by directing light into the eye and thus contracting the pupil. When the near acuity was the better, the distance acuity also could always be made better by the same method, and by the use of the pin hole

disk held before the eye. It is evident that vision is poorer when a contracted pupil shuts out a considerable amount of the direct rays of light from the object viewed, but vision may be improved when a small pupil cuts off scattered and disseminated rays. The former condition usually prevails with nuclear cataracts and those in which a small beam of light gives a sharper image.

Corneal Scars: Observations were made on many cases of corneal scars but only 186 of these were carefully tabulated, consecutive cases being used as far as possible. In these, the near acuity and the distance acuity were carefully recorded, as well as the age of the patient, density, nature, position and depth of scar. Only those cases having distance acuity between 6/7.5 and 6/60 have been used. Many small dense scars, one mm. or less in diameter, situated over the pupil, but not centrally, caused no diminution of acuity. These cases are not included in our data. It would take more space than has been allotted to me to discuss all these observations, therefore we shall give only those observations and conclusions which are most apposite to the subject.

Of these 186 cases, 29, or approximately 15%, had better distance acuity than near; 31, or approximately 17%, had better near acuity than distance; and 126, or 68%, had no difference in acuity. In those cases in which the distance acuity was the better, we found that the corneal scars involved the center of the cornea, were rather sharply defined, and were of uniform density, but they did not cover the entire pupillary opening. In those cases in which the near acuity was the better, we found that the scars were as large as the pupillary opening, that they did not have a uniform central density, and that they were associated with irregular astigmatism. In those cases where there was no difference in acuity, we found the scars were of uniform density and generally larger than the pupillary opening.

Considering corneal scars alone, there was found to be a difference between distance and near acuity in 22% of the cases; and considering all of the abnormal cases studied (850 cases, not including glaucoma), a difference between near and distance acuity was found in only 8% of our cases. Therefore, among scars of the cornea we find by far the largest possibility of a difference between distance and near acuity.

Surveying all cases having a positive difference in acuity,

we find the greatest variation to be in the ratio 1 to 2 or vice versa. These larger variations were found when acuity had fallen to a serious degree; for example between 6/18 and 6/60. This greatest difference in acuity was found in nuclear cataracts. The greatest difference in the perceptive faculty did not exceed 25% in any case, the visual angle being double, and in more than three-fourths of our cases, the difference was less than 5%, a difference of 2½ to 5 minutes of visual angle.

Deductions.

Altho, as pointed out by Professor Snellen, the distance test for acuity is more accurate than the near, nevertheless, the inaccuracies of the near test may be overcome by careful attention to details, especially by the use of a test card which conforms to the Snellen standard, and by careful determination of the near visual angle.

In determining a percentage loss of vision, such as is often demanded in medicolegal or in compensation cases, our investigations would clearly indicate, that in only those injuries in which the center of the cornea or lens has been involved is it necessary to make a near visual acuity test, since, in other cases of ocular disability, the near acuity and the distance will be identical.

In cases of opacities in the lens and in the cornea which are uniform in density and cover the entire pupillary space, the near acuity may be assumed to be identical with the distance without special testing. In general, those cases, especially irregular and central corneal scars and also nuclear cataracts, in which a distance acuity is increased or decreased by a change in size of pupil should be carefully tested for their near acuity as well as distance.

Since scarring of the cornea is by far the most common sequel in industrial accidents, and since in this class of cases there was found the largest number which had a difference between the near and the distance acuity, a careful near test as well as distance becomes imperative, in order to determine accurately any loss of vision at the working distance.

Summary.

In addition to variations due to the personal factor in any subjective test of visual acuity, the near test is less accurate than the distance, since the same measured errors

of size and of print are many time greater for near than for distance, and the rate of increase in error becomes increasingly greater as the objects approach the eye.

Near acuity should be expressed in the same manner as distance acuity, in conformity with the Snellen formula, in which the numerator indicates the distance at which the test is made and the denominator the distance at which the letters subtend a five minute angle.

When the distance of the punctum proximum, optimum, and remotum are all expressed in the numerator, and when the denominator expresses the distance at which the type used in the test subtends a five minute angle, a convenient, accurate and simple method is employed which reveals power of accommodation, the available region of accommodation and the acuity. This method conforms with the Snellen formula.

The term diopter can in no sense be applied to size or sequence of near test type. The designation "D" on many test cards properly referes to distance. The numerals connected with the "D" on many test cards properly refers to distance. The numerals connected with the "D" indicate the *distance* at which the letters subtend an angle of five minutes, or the *distance* at which an eye of standard acuity should be able to read the print. Errors of interpretation would be avoided if these distances were always expressed in meters or decimal fractions thereof.

The visual angles for best reading ability and for best acuity are not identical. The former is always the larger and coincides with the punctum optimum, whereas the latter is identical with the punctum remotum for the size of test letter employed in the test.

Acuity of vision at near points and at distant points is identical for normal eyes when the test is properly conducted, due consideration being given to refraction, accommodation, illumination, and size of visual angles compared.

Acuity at near and at a distance is also identical in eyes having subnormal acuity, except in those cases in which the size of the pupil may influence the acuity. Whenever the distance acuity, as measured under normal conditions of testing, is improved, either because of a contracted pupil brought about by indirect light stimulus, or by the use of a pin hole disk, the near acuity also will generally be found to be better than distant. But when distance acuity is made worse by

READING CARD (Metric)

4

I was dirty from my journey, and I know no soul nor where to look for lodging. I was very hungry, and my whole stock of cash consisted of a Dutch dollar, and about a shilling. I walked up the street, gazing about, until near the market house I met a boy with bread. I had made many a meal on bread, and asked him where he got it. I then went to the baker's and asked for biscuit such as we had in Boston. I asked for a three penny loaf and was told that they had none such.

5

Not knowing the difference of money and the greater cheapness, I bade him give me three penny worth of any sort. He gave me three great puffy rolls. I was surprised at the quantity but took it, and having no room in my pockets, I walked off with a roll under each arm. Thus I went up Market Street as far as Fourth Street, passing by the house of Mr. Reed, my future wife's father.

6

She saw me and thought I made a most awkward appearance. Then I turned and went down Chestnut Street and a part of Walnut Street, eating my roll all the way, and found myself again at Market Street wharf near the boat I came in. Being filled with one of my rolls, I gave the other two to a woman and her child.

7

By this time the street had many clean and well dressed people in it, all walking the same way. I joined them and was led into the great meeting house of the Quakers. I sat down among them and after looking around a while and hearing nothing said, I fell fast asleep, and continued so till the meeting broke up.

8

This was the first house I was in, or slept in, in Philadelphia. Looking in the faces of people, I met a young man whose countenance I liked, and asked if he would tell me where a stranger could get lodging. "Here", said he, "is one place, but it is not a reputable house."

9

"If thee wilt walk with me, I will show thee a better." He brought me then to a place in Water Street, where I got dinner. While I was eating it several sly questions were asked me, as it seemed to be suspected that I might be some runaway.

10

Then after dinner, I lay down without undressing and slept till six in the evening. Our city, though laid out with beautiful regularity, had the disgrace of allowing its wide and straight streets to remain long unpaved.

11

In wet weather the wheels of heavy carriages plowed them into a quagmire and in dry weather the dust was offensive. I saw with pain the inhabitants wading in mud while purchasing their provisions.

BAUSCH & LOMB OPTICAL CO., ROCHESTER, N. Y.

The above letters subtend the visual angle of 5′ at the distance designated in decimeters.

H-577

contracting the pupil, the near acuity will also generally be found to be poorer than the distance as determined under normal conditions of testing.

Our observations show that the proportion of cases of abnormal eyes in which there could be found any positive difference in the acuity at near and at distance are not numerous —8% in 850 cases—and when any difference was found, it was of low degree in a large percentage of cases. Altho any difference in relative acuity for near and for distance, when found, was of low degree in that class of cases in which industrial accidents are likely to cause partial permanent ocular disability, nevertheless differences do occur in some proportion of these cases, so that in the interest of aiding in the exact determination of visual loss, the near test of acuity should be made with the same meticulous care as is the distance.

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DISCUSSION.

Dr. Nelson M. Black, Milwaukee, Wis.: I wish to express my appreciation of Dr. Snell's excellent paper, especially that portion referring to near vision acuity. This is an item in the examination of the eyes to which we, as practicing ophthalmologists, are prone to give too little consideration.

It was not until suggested by the Committee on Compensation for Eye Injuries of the American Medical Association, that near vision was considered of sufficient importance to be used as a factor in estimating the visual efficiency of an individual. Near vision was deemed of such importance by the Committee, that it was given a valuation in estimating visual efficiency of 2-1 over distance vision.

The author, in his summary, states that "the near test for visual acuity is less accurate than the distance." I wonder if this is really a fact. True, one is able to detect slight differences in size and defects in contour when objects of sufficient size to subtend the visual angle are viewed close at hand, but is not that the result of the increased ability to observe detail, which is impossible when objects of visual angle size are viewed at a greater distance?

I feel sure we all will welcome a method of recording visual acuity for near that will be uniform, and will convey the same meaning to everyone, as does V = 6/VI or 20/20. By noting near V. A. as a fraction of which the numerator gives the distance at which the test characters are best seen, and the denominator the distance at which they subtend the five minute angle, we have something which is not only definite and uniform, but which is scientific and corresponds with our method of recording distance visual acuity; in addition, the suggestion of expressing in the numerator the distance of punctum proximum, optimum and remotum, as stated by the

author, shows in one record and at a glance the visual acuity, the power of accommodation, and the available region of accommodation or range of useful acuity for near.

Dr Snell's observation that there is a difference (varying as much, in some instances, as 50 per cent. in the size of the visual angle) in the test for best visual acuity and the ability of a person to read, is most interesting; also, that for the purpose of accurate determination of best V. A. for near, it is necessary to confine the test to single block capitals at the remotest point at which they may be identified. This brings us more and more in line with testing for distance acuity, and makes for uniformity in our tests, provided always that we use test charts that are correctly made to correspond to the five minute angle under proper illumination. It would also seem to me, that from the viewpoint of industrial visual acuity, single block capitals used for the test will give a better idea of what the individual's vision actually is, as in his work he is not attempting to interpret ideographs or printed words, but actual detail.

The question of size of the pupil influencing visual acuity is hopeless as far as uniformity is concerned, unless testing is conducted under uniform laboratory conditions.

The following reports of two cases seem to confirm the author's observation, that in industrial accidents causing partial permanent loss of vision, the difference in relative acuity for near and for distance is of low degree, but of sufficient amount that it is a factor in estimating visual efficiency. Hence the near test for visual acuity, especially in medicolegal cases, should, as Dr. Snell advises, be made with the same meticulous care as is the distance test.

E. L. W. March 2, 1925, penetrating injury to O. S. F. B. removed thru wound of entrance. February 4, 1926, media clear. Large patch chorioretinal atrophy down and out, extending into macular region—result of injury. No contraction of peripheral fields. No diplopia in any portion of field of binocular fixation. Absolute scotoma includes central fixation. Vision O. D.; distance, =20/15; vision near =14/14=100 per cent. Vision O. S.; distance 20/200=20 per cent. V. A. 14/178=12.8 per cent.

A. W. O., struck by auto January 2, 1926; injured about head, causing retinal hemorrhage O. S. in macular region. February 25, 1926, no contraction of peripheral field; no diplopia; dumb bell shaped absolute scotoma O. S. includes central fixation and blind spot. Vision O. D. =20/15 = 100 per cent. V. A. Vision O. S. = 20/200 eccentrically = 20 per cent. V. A. Near vision O. D. = 14/14 = 100 per cent. V. A. O. S. = 14/178 = 12.8 per cent. V. A.

Dr. J. M. Patton, Omaha, Neb.: Dr. Snell has rendered us a valuable service in presenting this subject. While there is an unfortunate lack of uniformity in the method of recording our distance visual findings, the measurements are based on a test character subtending a five minute angle and observed at a distance of twenty feet, or six meters, so that the records are usually intelligible whether written in decimals, fractions, or numerals.

In near vision records, this is not the case. As emphasized by the author, there is no recognized standard of test character or recording formula. A few years ago, this was of little importance, but with the present physical demands in many competitive occupations, the near may be more important than the distance vision, and both employers and compensation boards have a right to demand a standardized test record which is recognized by our profession and all others making tests of visual acuity.

The notation of the punctum proximum, punctum optimum, and punctum remotum in the numerator of Dr. Snell's formula preserves a graphic record of the patient's near visual acuity, and altho in the majority of cases observed, the relation between distance and close vision was constant, the record is none the less valuable. As the greatest variation was found where there was disturbance (often traumatic) of central corneal and lens transparency, the value from a medicolegal standpoint is the more apparent.

Dr. Snell, after careful observation and research, has devised a practical and accurate method of record, and I sincerely hope that the members of this organization will accept the author's suggestions. None can question the need of an established standard, and the influence of the Academy's endorsement would insure its general adoption.

Dr. Edward Jackson, Denver, Colo.: In the very large amount of important matter that has been presented by Dr. Snell it is quite possible that a very important practical point may be overlooked. It is very well stated in his abstract in our program in this sentence: "Many expressions for near vision are inaccurate and meaningless." The most common expression is "Jaeger" some number. If you have a copy of Jaeger's original test type, that may be done, but there have been scores of reproductions put out as Jaeger test cards, based on different forms of type, so that the commonest expressions with reference to near vision in the literature, or that we encounter in the case records which are brought before the Board for Ophthalmic Examinations, are absolutely valueless as an expression of any scientific fact.

I think the principles Dr. Snell has laid before us are sound and comprehensive, and the method he suggests is one that should be considered very carefully. From this point of view, this is an extremely valuable communication.

Dr. J. S. Lichtenberg, Kansas City, Mo.: I simply want to call attention to one point in the numbering of the test cards. The one marked with the number over the type in decimals similar to diopters is taken mistakenly by many to indicate loss of accommodation at the near point in diopters and is supposed to indicate the number of diopters of loss of accommodation, and to be added to the emmetropic or ametropic eye to the amount indicated by the number of even the smallest type the patient is able to read at ⅓ meter—either presbyopic loss or otherwise. That is a mistaken idea on the part of many.

Dr. Albert C. Snell, Rochester, N. Y. (closing): Dr. Black brings up the question whether the near card is less accurate. I think that ought to be obvious to all. It is not less accurate if you conduct the test in the way I have indicated, and especially if you will use block letters and not small case type.

The last speaker has brought a fact to my mind, and that is that there is no test that is perfect.

The four cards, those having block letters, those having small case letters, those having illiterate characters, and the cards in decimeter scale, are now published by Bausch and Lomb and may be obtained from them.

The expression for notating near acuity is not new, and the method is familiar to everyone in testing distant acuity when it is necessary to bring the distant card nearer than twenty feet, for those cases with acuity less than 20/200.

PRACTICAL APPLICATION OF ORTHOPTIC AND FUSION EXERCISES IN SUBNORMAL ACCOMMODATION, AMBLYOPIA, AND STRABISMUS.

OTIS WOLFE, M.D.

MARSHALLTOWN, IOWA.

To obtain and maintain efficient and comfortable binocular vision, we must have fusion. There must be a simultaneous nerve impulse coordinating the extrinsic muscles and causing them to adjust themselves so that rays of light can fall on corresponding areas of the retina. We must also have an equal accommodative effort, so that when rays of light focus on the retina, equal nerve impulses will be transmitted to the brain cortex. The efferent nerve impulses stimulating convergence will then be in their proper proportion. Factors that interfere with this are numerous and diverse. Refractive errors and heterophoria are important elements, but as we study anomalies of vision and asthenopia, especially in children with subnormal accommodation and amblyopia, we are impressed with the fact that other elements need our attention. The psychic and mental element of vision is the most important, but is the least understood. It is like electricity. We know its manifestations, its utilization and control, but its complete analysis is difficult to explain.

We can give much study to the subjects of perspective (Maddox[1]) projection, and ocular orientation (Duane[2]); also to the numerous phases of retinal stimulation, the neuromuscular reflexes, the gradual increase and decrease of visual sensation, and to retinal and cerebral stimulation and fatigue. The case reports will demonstrate their clinical aspect. Examination of the visual fields with the perimeter and the campimeter will map out pathologic areas of the retina, but we find a number of cases, especially in children, in which the condition seems to be largely a lack of development. In these cases, the vision of the two eyes may be equal or nearly so, and while no pathology can be demonstrated, there is little or no improvement with glasses. In unequal eyes, binocular

vision is not maintained. The poorer eye suspends vision partially or intermittently, and this condition is termed suspenopsia. If the condition is exaggerated until little or no vision exists, it is called amblyopia ex anopsia (amblyopia of nonuse). As we study suspenopsia and its relation to asthenopia, we are impressed with the great number of cases which lack the ability to maintain comfortable binocular vision and fusion if the eyes are subjected to much use. This is particularly apt to occur if the refraction or accommodation of the two eyes differ, especially if the patient is debilitated. Testing or treating fusion by means of rotary or loose prisms is unsatisfactory, due to the frequent presence of suspenopsia.

If we are to obtain other than a cosmetic result in the correction of strabismus, we must develop some degree of binocular fusion. It is doubtful, unless some degree of fusion exists primarily, that it ever can be developed. In the majority of cases, it has existed to some extent and can be restored partially or wholly. There are many factors that cause or influence heterophoria or heterotropia, and all must be studied out. Errors of refraction in the two eyes are of great importance and must first be corrected. No child is too young to have this done with cyclopegia and retinoscopy. In many cases, this will suffice to correct the fault. If the visual axes are not parallel and have remained so for a considerable length of time, fusion exercise alone is not apt to succeed. It is our rule, however, if the strabismus does not improve after cyclopegia, that the case is not one caused by excessive convergence and accommodation alone, but that anatomic factors are present which necessitate operative treatment also.

It is probable that most cases of comitant convergent strabismus were alternating in their inception. As we study alternating strabismus, we find many of them with normal vision or slight refractive errors when either eye fixes (case No. 1). Binocular fusion cannot take place if anatomic factors are present so that the eye cannot fixate and effect simultaneous retinal stimulation (central fixation) (case No. 2).

Hyperphoria is very often present and should carefully be considered.

In a study made of the fusion ability of children without strabismus, ranging from four to ten years of age, I have found faulty fusion in many of them, and many others in which it is imperfectly developed. In a series of cases studied

at the Iowa State Training School for Boys, we found twenty-seven boys who had weakness of fusion but no asthenopic symptoms. In checking over my refractive records of patients with asthenopia, I found eighty-seven adult hyperopes who showed absent or imperfect fusion at the first examination, with little or no muscular error. Subnormal accommodation frequently accompanies it. We find a large number of hyperopes, both in adults and in children, with convergence insufficiency and pronounced exophoria at the near point. This is probably due to weakness of fusion and subnormal accommodation. There is a wide range in fusion power. Some adults as well as children cannot fuse small objects, but will fuse readily large ones. Some cannot fuse readily after the eyes have been subjected to considerable close work. A wide variation is found in the amount of stereoscopic fusion ability. Refinement and development in quality of the fusion faculty is accomplished by the Wells charts and other exercises mentioned. We also develop the amplitude of fusion convergence, so that these patients can continue to use their eyes at near work with comfort.

Treatment of Amblyopia ex Anopsia and Functional Amblyopia.

The methods commonly used to overcome amblyopia are to bandage or atropinize the good or fixing eye. This is good as far as it goes, and it should be utilized to some extent, but it utterly ignores binocular fixation and fusion. It demonstrates its deficiency, if persisted in, by oftimes converting the fixing eye to the converging one. It does, however, serve the purpose of demonstrating that most or all comitant convergent strabismus are alternating in their inception, and the lack of the fusion element. In dealing with the functional amblyopia of children and young adults, we must also develop perspective, projection, and stimulate the retinal fields, as well as endeavor to restore binocular fixation and fusion in the phorias and the tropias. If suspenopsia exists, as it very frequently does, we must develop and train the secondary visual centers. In strabismus, we must also deal with the complicated mechanism existing between these factors, accommodations, and extrinsic muscles.

The Worth amblyscope has a distinct place early in developing these cases, but the Wells system of stereoscopic ex-

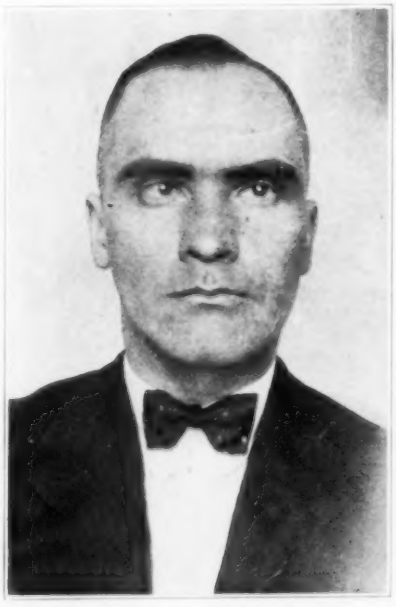
ercises with the phorooptometer will do more to stimulate all of the desired elements. The strong lens of the stereoscope hold accommodation in check while fusion is incited. Wells[3] has elaborated on the works of Javal and others. The perspective element, which is very important, is given special attention. The brain views images much like the stereoscope; the retinal pictures are blended into one image. The visual act is a threefold affair, muscular, physiologic, and mental, but largely mental. In utilizing the Wells charts, we are able to stimulate the mental process of vision, while at the same time we can keep an accurate record of the amount of stimulation that we are obtaining in the visual centers of the brain. The aim of stereoscopic exercises is to restore, first, binocular vision, and then if possible develop stereoscopic vision. We cannot have perspective if suspenopsia exists, and therefore stereoscopic vision cannot be obtained. The existence of suspenopsia is immediately noted when using the Wells charts. Prism exercise with the Hazen krotometer will accomplish it, but as it is quite complicated, it is almost necessary that it be operated by the oculist himself.

In conjunction with the Worth amblyscope and the Wells stereoscope, I wish to submit for your consideration an exercise that I have not seen described elsewhere. It is easily carried out, and if persisted in, it seems to have a far reaching effect. All other exercises for fusion are at the near point, where accommodation is bound to be utilized to a more or less degree, which is admittedly undesirable, especially in convergent strabismus. The exercise is as follows: After the good eye has been covered for about an hour, it is unbandaged. A Snellen chart or picture chart for children is placed where illumination is good. A piece of cardboard is held before the good eye, and the patient views the smallest line of letters he can plainly read at a distance of ten feet or more. He is told to move slowly backward, keeping his vision fixed on the letters. As soon as he loses the letters, he is instructed to step forward until he catches them again, and then to go slowly backward. Each time the letters begin to blur, he uncovers the good eye for a second and immediately recovers it. He must be cautioned to be fixing with the poor eye when he uncovers the good one. This is repeated several times. When he is fatigued, the eyes are rotated in a circle

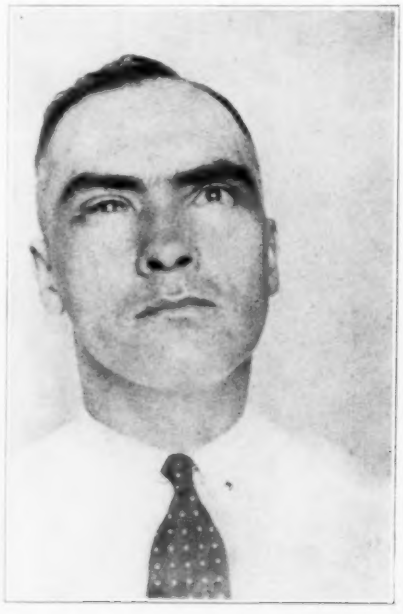
and the above process repeated. Children are encouraged to study the relative size of the pictures and of the letters in the different lines. With this procedure, we are utilizing perception, projection, perspective, and are stimulating binocular fixation and fusion with a minimum of accommodation and convergence. For amblyopia of convergent strabismus, the correcting lens should be worn. Carefully regulated atropinization or partial fogging of the fixing eye are useful in difficult cases. In hyperopes with divergent strabismus, accommodation and convergence should be stimulated as much as possible, i. e., exercise begun at near point with the correction left off. The above exercise also has a beneficial influence in certain cases of myopia.

Case Reports.

Case 1. Mr. C. J. (Fig. 1), age 30, clerk. Alternating strabismus since early childhood. Never could control eyes, and chagrined that he could never tell which one was fixing. Exam.: Vision 18/15 both eyes. Would accept only sp. plus .25 and cyl. plus .25, both under cycloplegia. Shows 25 degrees convergence. No fusion present. Operation: Advanced right

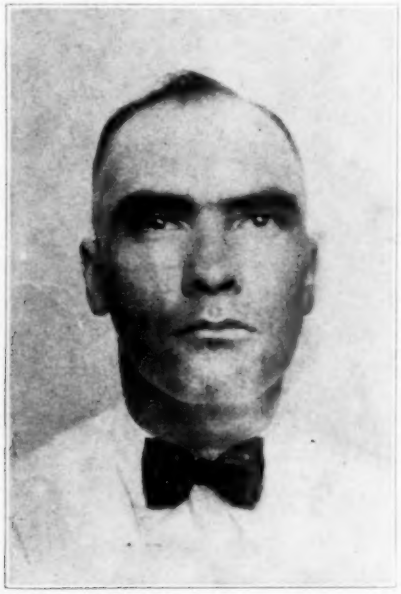


Case 1. Mr. C. J. Picture 1



Case 1. Mr. C. J. Picture 2

external rectus. Picture No. 2 taken next day. Fused lights at 20 feet. Maddox test shows orthophoria at near point. With stereoscope, fuses F and L to E but not small letters. Could fuse up to G5 (Wells). One week later, fuses small letters in chart C8 and G8. Did Herring drop test two weeks after. He returned to his office work three days after the operation. Picture No. 3 taken three weeks after the operation. No glasses worn.

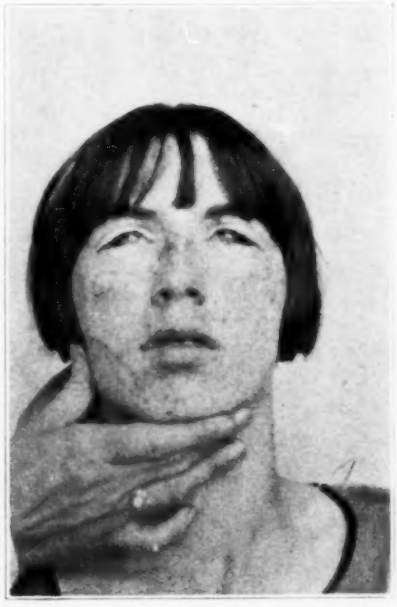


Case 1. Mr. C. J. Picture 3

Case 2. Irene H. (Figs. 4 and 5), age 16. Eyes began to cross when four years old after whooping cough. When ten years old, effort made to straighten with glasses without success. Strabismus has always alternated, but tendency now for right eye to fix. Vision, right=20/20; left=20/20. Under cyclopegia, shows plus 2.00 sp. both eyes. Ten degrees accommodation in each eye, but no fusion present. Intermittent suspenopsia left eye shown with stereoscope. 35 degrees deviation when fixing with right eye. Operation: Advanced right externus 20 degrees and left 15 degrees under local anesthesia. Next day (Fig. 6A) fused lights at distance, F and L stereoscopically, but not small objects. Two degrees hyperophoria present, but this disappeared in ten days, when

Case 2. Irene H. Picture 4

Case 2. Irene H. Picture 5



Case 2. Irene H. Picture 6A

Case 2. Irene H. Picture 6B

she showed 2 degrees esophoria at distance. Fusion and stereoscopic exercises. Six days after the operation, she could fuse small letters G1 to G7. Two weeks later, she could do the full series. Herring drop test positive. (Fig. 6B). Six weeks after operation, vision and accommodation were normal Glasses discarded.

CASE 3. Eva S. (Fig. 7), age 13. Right eye had been crossed since three years old. Fell off porch; struck right

Case 3. Eva S. Picture 7

forehead. Treated with glasses five years ago. Wearing right sp. plus 5.00, left sp. plus 3.50. No improvement in the squint. Right comitant convergent strabismus 35 degrees. Vision right 20/200, vision left 20/20. Under cyclopegia, former correction found to be O. K. Vision improved only slightly with glasses and strabismus not at all. Advised immediate operation, but parents desired effort be made without. Worth amblyscope and Wells stereoscopic exercises for two months. No improvement. Operation: Advanced right external rectus 35 degrees under local anesthesia. Picture 8 taken next day. Back to school second day without dressing. Fused lights at distance and F and L to E with stereoscope.

Case 3. Eva S. Picture 8

Case 3. Eva S. Picture 9

Case 4. Leo P. Picture 10

Case 4. Leo P. Picture 11

Has never been able to go above G5 card. No depth perception. Herring test negative. Picture 9 taken one year after.

CASE 4. Leo P. (Fig. 10), age 9. Eyes have been crossed since infancy. Strabismus 35 degrees. Right eye fixes but left eye can also be made to fix. Attempt was made by an oculist to straighten with glasses when four years old, but no fusion exercises were given. No result. Exam.: Vision right 18/30; vision left 18/100. No fusion. Under atropin

Case 4. Leo P. Picture 12

cyclopegia took sp. plus .50 ⊃ cyl. plus .75 axis 90 both eyes. This did not improve his vision, but strabismus was ten degrees less. With fusion and orthoptic exercises over a period of several months, his eye gradually became straight. Picture 11 and 12 one year after. Vision left 18/30, vision right 18/20. No operation. Suspenopsia of left eye was probably the reason the first treatment with glasses failed. Positive Herring drop test.

CASE 5. Mrs. S. (Fig. 13), age 39. Developed convergent strabismus after severe attack of pneumonia. Exam.: Vision right 18/30; vision left 18/30. Refraction shows sp. plus 50 ⊃ cyl. plus 25 axis 180; gave 18/20 both. Convergent non-

Case 5. Mrs. S. Picture 13

Case 5. Mrs. S. Picture 14

Case 5. Mrs. S. Picture 15

comitant strabismus varying from 30 to 45 degrees. Right had shown iridoplegia for several years. Wassermans, both blood and spinal, were negative. No fusion present. Accommodation weak and varied in right, left 3 D. Advancement advised, refused. Prisms prescribed; fusion exercises with internal medication continued at intervals over a year. Eyes restored to normal. Right iridoplegia and subnormal accommodation remains (Figs. 14 and 15). See Duane[4] on static

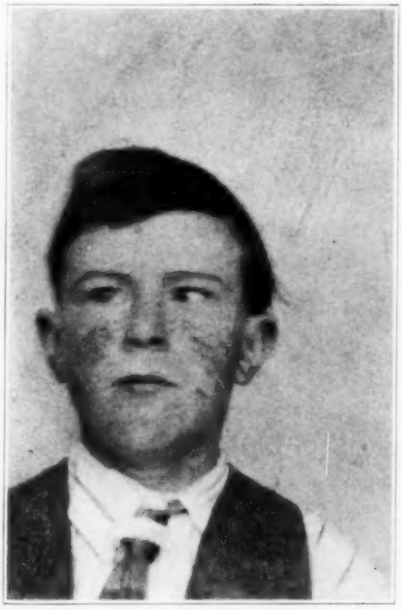
Case 6. George N. Picture 16

subnormal accommodation with iridoplegia. Could do Hering drop test at times.

Case 6. George N. (Fig. 16), age four. Left eye crossed since five months old. Exam.: Convergent strabismus 30 degrees. Vision right 12/30; vision left fingers two feet. Atropin and retinoscopy gave right sp. plus 2.50 ⊂ cyl. plus .50 axis 90. Left sp. plus 3.50 ⊂ cyl. plus .75 axis 60. Did not improve the strabismus. No fusion. Advanced left external rectus under general anesthesia. Very small atrophic muscle, attached well back on globe. Picture No. 17A taken three days after the operation. Dressing worn only one day. After five months he could fuse large objects well, and small

Case 6. George N. Picture 17A

Case 6. George N. Picture 17B



Case 7. Harry S. Picture 18

Case 7. Harry S. Picture 19

ones with difficulty. (Fig. 17B). Eyes straight. Vision with his glasses, right 20/20 and left 20/20 minus.

CASE 7. Harry S. (Fig. 18). Age 9. Left eye crossed when three years old. Treatment with glasses when six years old, and again when eight. No result. Exam.: Vision right 18/15, left 18/100. Wearing right sp. plus 2.50, left sp. plus 2.00, which corrected his vision to 18/70. Under cyclopegia, found right sp. plus 2.00 and left sp. plus 1.50 ⊃ cyl. plus .75

Case 8. Agnes R. Picture 20

axis 90, corrected to 18/40, but strabismus did not decrease. No fusion. Strabismus was noncomitant, varied from 30 to 40 degrees before and under cyclopegia. Advanced left external rectus under general anesthesia. Found atrophic muscle attached way back on eyeball. Picture No. 19 taken next day shows eyes straight. Fused light at distance, but not with stereoscope or with Worth amblyscope, due to suspenopsia of left eye. One month later, he could fuse all large objects and some small ones. Series G (Wells) could be fused as far as number 5. Herring drop test positive.

CASE 8. Agnes R. (Fig. 20). Age 17. Eyes turned out since early infancy. Asthenopia; had treatment with glasses

by an oculist for one year. No improvement. Exam.: Vision right 18/70, vision left 18/30. Refraction right eye sp. plus .50 ⊃ cyl. plus .50 axis 90, gave 18/30. Left eye sp. plus .50 ⊃ cyl. plus .50 axis 90, gave 18/15. Abduction 11; adduction 22. Can fuse lights and large stereoscopic pictures for short intervals with effort, but right eye suspended vision most of the time. Strabismus alternated. Right eye shows 15 degrees deviation outward, and the left ten degrees outward. Diag-

Case 8. Agnes R. Picture 21.

nosis, alternating divergent noncomitant strabismus with hyperopia. With Maddox tangent scale near point 38 cm. Shows 15 to 20 degrees convergence insufficiency. Fusion exercises for one month, then advanced right internal rectus. Picture 21 taken two weeks after operation. Could maintain stereoscopic fusion of F and L on C8 chart but not small letters. In three days, good fusion for small letters, had stereoscopic vision and depth perception, but must continue exercise to maintain it. Hyperphoria immediately after operation, but cleared when tissues adjusted themselves.

I wish to mention briefly some points regarding the dynamic type of subnormal accommodation without iridoplegia

(functional insufficiency), and point out their practical value in a couple of case reports. For details I would refer you to the excellent work of Dr. Duane,[5] where he classifies the different types and goes into the subject in his usual thoro manner. He stresses bar reading. The clinical application of Duane's work will enable the oculist to dispense with glasses in many cases. His table of accommodation should be generally studied and used. I wish to emphasize, however, that glasses should be dispensed with only where the refractive error in the two eyes is equal, or if the accommodation is equal or can be brought up to normal by exercise, tonics, hygienic measures and by the elimination of focal infection.

Nasal obstruction and sinusitis has been demonstrated to be a positive factor in several cases.

Eunice T. Age 9. Came under our attention 4 years ago. Parents noted she held reading very close and had headaches after she used her eyes for close work. Exam.: Vision right 18/40, vision left 18/40. Ophthalmometer showed 2 degrees astigmatism at vertical axis. Refraction under atropin and retinoscopy gave right sp. plus 2.25 ⌒ cyl. plus .50 axis 90. Left sp. plus 2.25 ⌒ cyl. plus .75 axis 90. .50 sp. was deducted from the prescription. Glasses gave complete relief. Parents then wanted to know if anything could be done so she would not have to wear glasses. Exam.: Pupils were rather large and reacted slowly to convergence. Two degrees esophoria at distance, no diplopia. Four degrees exophoria at 30 cm. Convergency insufficiency with Maddox test. Accommodation right 7 D. and left 6.5 D. Could not fuse F and L on chart C8 (Wells). Fusion and convergence exercises above described, also bar reading over a period of four months. Accommodation (Duane's table) came up to normal, 14 D. both eyes. Vision right 18/15, left 18/15. Tonics administered. Glasses were dispensed with entirely. She has gone to school and had excellent grades with no symptoms whatsoever. One year later, acc. 16 D.

Katherine A. Age 11. Came to us three years ago. Had frontal headaches after use of eyes. Could not see work on blackboard at school. Given glasses by oculist who used cyclopegia. Our exam.: Vision 18/200 both eyes. Was wearing sp. plus 2.00 ⌒ cyl. plus .75 axis 90, both eyes, which gave her 18/50 vision. Ophthalmometer showed 1.75 cyl. ver-

tical axis, and would accept 1.25 in both eyes. Total correction and prescription sp. plus 2.00 ⊃ cyl. plus 1.25 axis 90 both, but vision would not go above 18/40. 4 D. exophoria at distance. 10 D. exophoria at 38 cm. (convergency insufficiency, Maddox). Accommodation could not be estimated, as vision was not acute enough. No stereoscopic fusion. Under exercise mentioned above, after six months vision right 18/15, vision left 18/15 without glasses. Fuses G10 (Wells) with slight difficulty. Her general health had been below par. Tonics were given. She is the picture of health now and full of vitality. She has not worn glasses for two years, even for close work. No symptoms.

After this paper was completed and copies sent to Dr. Jackson and Dr. Wells, the patient returned to me for reexamination. She lives in a distant state. I first saw her two years ago, and my case report was based on the findings of one year ago. On account of complete absence of symptoms, she had discontinued her exercises.

Our exam. August 22nd: Vision right 18/30, vision left 18/30. Accommodation 8 D. both eyes. Her astigmatism, however, was .75 D. less in both eyes. Excellent health. No asthenopia. I advised intensive exercise, and that glasses be worn for close work or until the accommodation comes back to normal. This rather extreme case demonstrates the limitations of treatment as well as its possibilities.

We have, however, many cases who have been able to discard glasses completely after short periods of treatment.

I[6] reported two other interesting cases of late fusion development in the American Journal of Ophthalmology, January, 1927. One was a case of convergent strabismus since early childhood in a man 47 years old, in whom we were able to develop fusion. Herring drop test positive.

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DISCUSSION.

DR. EDWARD JACKSON, Denver, Colo.: There is a phase of this subject that is of great practical importance, and that is overlooked largely from confusion of ideas that arises from using habitual phrases.

These troubles are very often spoken of as troubles of muscles. The difficulty is in the very accurate coordination of certain delicate movements. That is the trouble we are trying to combat, and we will get a better view, a more competent view of the subject, a view that will be new to some, if we compare the way we have been seeking to secure such coordination of movement of the two eyes with the ways that we generally use to get accurate coordination of our different movements. For instance, there are some in the Academy who will immediately think how they have developed a better game of golf; there are other instances of work of musicians. How do we get accurate coordination—not only strike the right key but with the right strength? How does a musician using a violin get the right coordination? Do we try to secure a strengthening of these muscles by operation? Do we seek accurate measurements of the different muscles to be used before attempting to get the ideals aimed at? If we will think of it in that way, we can all see that this matter of fusion training—one name given for it—is a development of coordination of the different impulses sent out from the nerve centers, and this development of coordination is to be sought more as we seek coordination of other muscles than has been usually tried for the eye muscles. And it is peculiarly necessary for the eye muscles. They are of small size, and the majority of the movements to be executed have very close association with cerebral actions of various kinds; and when it is investigated thoroly and weighed carefully, we will find, what I think a good many have already believed must be so, that we are dealing with a series of nerve actions.

Now that does not exclude at all the benefit to be obtained by operation. When these patients come to us, the nerve action, the faulty coordination, the improper coordination of movements, is an established habit—as much a habit as the faults which can be acquired by a piano player, or the wrong stance of a golfer—and the most direct familiar method of breaking that bad habit for eye movements is by operation. Every operation on the ocular muscles requires a new development of coordination; and the application, particularly at that time, of other methods of nerve training is, as the series of cases placed before us shows, very effective.

DR. A. DEH. PRANGEN, Rochester, Minn.: There is no doubt that orthoptic and fusion exercises are of definite benefit in selected cases of strabismus and amblyopia ex anopsia. Like any other form of training or exercise, however, careful supervision and direction must be given. In the absence of this, results will be very disappointing. The type of child that will cooperate in these exercises has been rather uncommon in my experience. Even more important than the child, however, is the cooperation of the parents. In spite of careful explanation of what is trying to be accomplished, it has been very difficult for us to get even the higher type of parents to put the exercises over. Both parents and children start out well, but soon the exercises are cast aside. This has been true even of such

simple a matter as occluding the sound eye for a part of each day. The cases in which we have had any success at all, are the ones which are able to come in for frequent office visits, so that we can check up methods, give directions and encouragement. In this more favorable group, we often have had very nice results. Parallelism of the visual axes and improvement in vision in the squinting eye have been obtained. Fusion is frequently reestablished.

Our efforts with the children have been confined chiefly to careful refraction and prescribing so as to balance the visual acuity. If the squint is alternating and the vision in both eyes is good, this is usually all that we feel necessary. If the squint does not improve or the vision in one eye is poor, then we resort to occlusion of the good eye and fusion training with orthoptic exercises where deemed practical. We have found the Worth amblyscope most useful for fusion training. For muscular insufficiency, exercise of the affected muscles, if carefully carried out and adhered to, are of distinct benefit. Merely correcting the error of refraction aids in preventing and also the overcoming of these insufficiencies.

Of special interest to me have been the children with squint associated with low errors of refraction. Almost without exception, the careful correction of the errors, small tho they may be, has caused immediate improvement to parallelism with return of fusion. Apparently the muscle balance of these children is very unstable and precipitated into a strabismus by the refractive error. Another similar interesting group, often encountered, are the children who develop an amblyopic eye unassociated with squint. These also have a small refractive error. There appears to be a suspension of vision in one eye which responds readily to occlusion of the good eye and exercise of the poor one, together with correction of the refractive error.

The improvement of a squint after cycloplegia is mentioned by Dr. Wolfe as a favorable prognostic sign. Such a case one would expect to be handled very well by correction of the refractive error. I think this is true. However, failure of a strabismus to improve under cycloplegia does not necessarily indicate that glasses will not improve the squint by use. Many cases not improved at the time of the cycloplegia, do improve with the use of glasses.

Perhaps the most important part of the problem of strabismus and amblyopia is the getting of these children to the oculist early. The medical profession, as well as the general public, too often have the idea that the unfortunate child will outgrow the difficulty. This is, of course, usually erroneous. It is this early neglect which causes most of the disastrous sequelae which are so hard to correct. I feel with Dr. Wolfe, that amblyopia ex anopsia is an acquired condition, and that fusion is lost by neglect or disuse. If these children could have immediate, early and proper attention, most of them would receive a prompt functional as well as cosmetic cure. Dr. Wolfe's paper will stimulate us to use orthoptic exercise and fusion training more often in cases of strabismus and amblyopia ex anopsia.

Dr. Otis Wolfe, Marshalltown, Iowa (closing): I was very much pleased with Dr. Jackson's emphasis of the mental element of vision. A great many children have to be taught to see.

Dr. Greenwood asked me how I estimated the degree I was advancing these muscles, which I said in one case was 35 degrees. I replied that I guessed at it and asked him how he did it. He said he did the same. We estimate that advancing one millemeter will equal 5 degrees on the perimeter.

We overcorrect them 5 to 10 degrees. It is like making love to a young widow—you can't overdo it.

A STUDY OF STANDARDS FOR JUDGING OF THE PROGRESS OR ARREST OF CATARACT.*

DANIEL B. KIRBY, M.D.

NEW YORK CITY, NEW YORK.

Having undertaken the study of the possibility of the prevention or arrest of senile cataract by nonsurgical means, it was found that it was necessary first to set up standards for judgment of results.

We saw that we must formulate a set of conditions under which two workers could independently make their observations on the same patient and arrive at uniform conclusions.

One essential for a prognosis in regard to cataract is the knowledge of its usual course under the favorable and unfavorable conditions in which our patients live and use their eyes. This Jackson[1] has given us in a very comprehensive manner.

It was proposed to use this partly as a guide, and to add to it the knowledge gained thru the study and classification of a large number of cases with the aid of the slit lamp microscope, and to correlate these findings with the changes in refraction which are incidental to the development of the various types of lens opacities.

It was also proposed to analyze the various methods of examining partial cataract, with a view of standardizing as many as possible of the factors concerned. The tests selected by virtue of their practical value were: 1. The Test of Vision; 2. The Test of Refraction; 3. The Test with the Ophthalmoscope; and 4. The Test with the Slit Lamp Microscope.

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*This thesis is part of a report on work conducted by the author as Research Fellow for the American Academy of Ophthalmology and Oto-Laryngology. He is indebted to the Academy, to Dr. John M. Wheeler, and to an anonymous donor for funds necessary to carry on the investigation. He wishes also to express his appreciation of his association in private and hospital practice with Dr. Wheeler, and of the privilege of the use of the clinical material which forms the basis of this report.

Definition of Cataract.

Cataract in this thesis includes any opacity of the crystalline lens which can be detected with the ophthalmoscope. The final diagnosis of cataract rests upon the ophthalmoscope. Inasmuch as the meaning of the term is often distorted in the lay mind, its use is often better avoided in conversation with patients.

In an effort to make this study as practical and complete as possible, much that is commonplace has been introduced. For this, the author prays the indulgence of the kind reader.

The Test of Vision.

Tests of central color vision, light sense, near vision and fields for form and color are of great value in determining the function of the eye, but the test of central visual acuity will always be of great importance in judging of the progress or arrest of partial cataract. Conditions for repeated testings of vision at regular intervals in research work, or at subsequent visits of patients in clinical practice, should be standard and reproducible. A clinician may choose any sort of illumination which he has found thru practice to be effective, but for subsequent tests the conditions should not vary.

The following conditions were found to be of practical value in testing the eyes of patients afflicted with cataract. They can all be standardized.

1. Test Object: The standard Snellen test type charts with black (95-99% opaque) letters or numbers on white ground (79-80% reflection value). Charts are replaced as the reflection values fall, due to soiling or age. Black etched figures on white mat finish glass are excellent, either with reflected or transmitted light, provided the illumination be controlled.

2. Distance from Object: Distances of 20 feet or 6 meters are the most useful. Other distances may be employed, but corresponding charts should be used. Discrepancies may arise from using a chart unsuited for the particular distance.

3. Expression of Visual Acuity: Visual acuity may be expressed in terms of the distance used, either in the English linear system or in the metric system, but the decimal system will find more universal understanding. Visual acuity may thus be expressed, 1., .8, .5, .1, .01, and should always indicate the corrected vision after the test of refraction has been completed.

4. Illumination of Test Chart: Test charts are for practical purposes best illuminated by artificial light falling upon the chart, so that the chart is visible by virtue of the reflected light. The intensity of illumination must be controlled, standardized and reproducible. A survey of the intensity of illumination in various oculists' offices would probably disclose the fact that each one has a different intensity, and hence each one must form his own standard of vision. Standardization of illumination for various tasks in the industries is progressing, and oculists can render their patients a greater service by making the final test under the conditions in which the patient will use his eyes. Old eyes with certain types of partial cataract need greater illumination of the object than do young eyes. Eyes with incipient senile cortical opacities can use greater intensities than eyes with nuclear opacities.

Ferree and Rand[2] use 5 foot candles for their preliminary tests of vision of the average eye. They then graduate the intensity for various purposes.

An investigation of the intensity of illumination best suited for use by eyes with various types of lens opacities, as demonstrated with the aid of the slit lamp microscope, should prove of value in getting the fullest efficiency from these eyes, both at the time of the test and in the average use of them by the patient. The latter will naturally seek the intensity of light which is best suited for his particular eyes and occupation.

Convenient arrangement may be made to vary the intensity of the illumination in any particular test unit, either by moving the source of illumination to or away from the test chart, or by introducing a variable resistance rheostat, the latter, however, having the disadvantage of increasing the amount of red color in the light at the lower intensities.

The light falling on the chart should be uniform. Bulbs, frosted, preferably on the inside, assist in this, but uniform illumination can best be obtained by transmission thru a filter.

5. The Test Room: For practical purposes, patients should be tested under conditions in which they live and use their eyes. A test room with neutral tone walls and daylight illumination value of not more than 3 f.c. is suitable. Objects such as furniture should not intervene between the patient and the test chart.

The Test With the Ophthalmoscope.

The transparency or opacity of any medium is to be determined by the ratio of the transmitted to the incident light. Therefore the transparency or opacity of the ocular media in normal and abnormal states is best judged objectively with the ophthalmoscope. The final diagnosis of cataract rests upon the ophthalmoscope.

The test is best made with the reflecting instrument, using the plane or concave mirror, with the aperture at a distance of about 50 cm. from the source of light, and 33 cm. from the patient's eye. Self luminous ophthalmoscopes are useful with the +16 D or +20.D lens if they permit of a proper focus and graduation of the light.

Tests were made to determine, (1) the optimum range of intensity of illumination for judging the transparency or opacity of the lens and (2) the intensity of illumination obtained with the plane and reflecting mirror at the distances named above with various seasoned bulbs. The mirror was placed in the plane of the eye of the observer, and the light reflected onto a photometer test plate of mat opal glass having a reflection factor of 0.79. This test plate was located in the plane of the patient's eye. Both outside frosted and inside frosted bulbs of different wattage were tested. These tests are not complete. A large number of seasoned bulbs must be tested before a definite report can be made.

The bulbs with filaments exposed gave unsatisfactory tests, as they produced uneven intensities on the test plate. For the same reason, they are unsatisfactory for testing the transparency of the ocular media.

The lower ranges of light intensities were found best suited for the author's eyes for determining the transparency of the crystalline lens.

The range beween 2.5 to 4 foot candles was most useful for detecting (1) water splitting of sutures, which show as faint striae, dark in one position of the mirror and light or disappearing in another; (2) lamina separation, which appears as a faint cortical haze; (3) vacuoles, which show as minute light rings with dark center; (4) beginning nuclear sclerosis, which appears as a faint nuclear haze.

Replacements should be frequent enough to insure against any great difference between the new and the old bulb. For carefully checked work, inasmuch as the manufacturers are allowed a certain percentage of variation above and below

the wattage rated on the bulbs, the light intensity should be controlled by a variable resistance rheostat, so that identical and constant factors may be had.

A graphic record by pencil drawing should always be made of the appearance of a lens opacity for comparison at subsequent examinations. The same light intensity, mirror and distance should be used in making the comparison. The drawing should indicate the combined effect of the opacities if they are multiple. A schematic representation of the anteroposterior position of the opacities may be made. These observations and records are best made when the pupils are dilated.

The light sense and the visual acuity of different oculists undoubtedly vary. Each one has his own empiric standard of the transparency of the ocular media. It is desirable for purposes of comparison of records and for uniform results that some standards be adopted. An examination of the light sense, the visual acuity and the intensity of illumination used by a representative group of oculists might help in this respect.

The Correlation of the Test of Refraction and the Test With the Slit Lamp Microscope.

Many and startling changes in refraction are seen in the process of formation of certain cataracts. The author proposed to analyze the material available from association in private practice with Dr. John M. Wheeler. The first purpose was to consider the changes in refraction of the patient in connection with the detailed knowledge gained from the use of the slit lamp microscope in the particular case; 2nd, to learn the particular type of lens opacity which is most likely to produce the familiar lenticular myopia of incipient cataract; and 3rd, to learn of the average expectancy of changes of refraction in the different types of lens opacities.

The observations and reexaminations have all been made within the past three years by Drs. J. M. Wheeler, T. H. Johnson, Z. H. Ellis and the author. Some of the records of refraction date back to the time of Drs. H. D. Noyes and D. W. Hunter.

This study comprises the recognition and classification of the various types of lens opacities seen in a total of 7950 patients of all ages in the past three years (July 1924 to July 1927, incl.).

Not all of these patients have been examined with the aid of the slit lamp microscope. The use of this instrument has grown to be almost routine by the author, thru interest in the beautiful detail which it affords. In all cases of doubt as to a particular feature observed with the ophthalmoscope, the slit lamp microscope has been called into play.

In 945 cases, 11.9% of the total number of patients, lens opacities were detected. 407, or 43% of these, were male, while 538, or 57%, were female. None of these had any local nonoperative treatment, except a very few who received dionin drops in increasing dosage.

The decision of opacity always rested on the ophthalmoscope. Certain features, in themselves transparent, as for example vacuoles or water split fissures, cause opacities in the lens because of the difference in their indices of refraction.

The various lens opacities were listed according to their significance. In 136 patients, opacities of two different types were found, so that in the 945 cases, the various types of opacities occurred 1081 times. For example, if a certain case of congenital cataract showed isolated anterior subcapsular vacuoles, they were cross indexed, but the incidence of vacuoles in cases of senile cortical cataract was not cross indexed, as the vacuoles are part of the condition.

The incidence of certain important mixed types of cataracts is given separately, as for example the combination of posterior cortical opacity with pathologic nuclear sclerosis.

With the desire of making the study of practical clinical value, artificial divisions are avoided. Thus lamina separation and water splitting of sutures, whether occurring alone or together, are listed under the clinical heading of incipient senile cortical cataract. Each eye of each patient is regarded singly, as a certain type of lesion may appear only in one eye, and, again, the two eyes of one patient may show different types of lens changes.

The Test With the Slit Lamp Microscope.

The use of the slit lamp microscope has become constantly of greater value as an aid in the practice of ophthalmology. This is particularly true in the determination of the exact nature of lens opacities which have been detected with the

ophthalmoscope. It is also of value as an additional test of the progress or arrest of cataract. Progress of most lens opacities is slow, so that sufficient time has not elapsed since the introduction and use of the slit lamp microscope for the gathering of sufficient data on which to base a prognosis in a given case. The knowledge gained by its use, however, promises much aid in this respect. At the present time, the stationary types of opacity can be distinguished from the progressive. The average rate of progress of the various types of progressive lens opacities is a matter for future research.

Many features which are visible with the aid of the slit lamp microscope are invisible with the aid of the ophthalmoscope. Features such as suture lines, nuclear relief, various coronary and axial strictures are visible by direct illumination, while they are invisible by transmitted or retroillumination. With these we are not particularly concerned in this study. The cases listed in this series include only those which could be detected with the aid of the ophthalmoscope. Faint capsular iridescence and minute punctuate and linear markings, described by Vogt as borderline between physiologic and pathologic conditions of the lens, were frequently observed but have not been listed in this series.

Special Technic.

A pathologic feature found in the crystalline lens may be examined by the following methods. The terminology is after Graves.[3]

Method	Special Use
1. Direct Illumination	(for direct observation of all opacities)
2. Retroillumination	(assists in judging transparency)
3. Marginal Illumination	(useful for detecting vacuoles)
4. Specular Reflection	(for demonstrating iridescence)

Localization or Judgment of Depth.

This is important in the determination of the chronology of the development of the various opacities. The narrow slit beam is most useful for this purpose. With its aid, one may localize and determine the depth of any opacity in reference to the principal planes of discontinuity.

Planes of Reference.

These are planes or zones of discontinuity due to variations of relucency of the lamina of every lens. The principal planes are.

1. Anterior and Posterior Capsular.
2. Anterior and posterior Surfaces of Adult Nucleus (localized by the nuclear relief).
3. Anterior and Posterior surfaces of the Fetal Nucleus (localized by the position of the anterior and posterior "Y" sutures).

These planes divide the lens conveniently for clinical purposes into: 1. Senile Cortex—developed after puberty—youngest fibers most peripheral. 2. Adult Nucleus—from birth to puberty. 3. Fetal Nucleus—prenatal period.

Observations on the condition of the vitreous and of the presence and number of pigmented deposits on the endothelium of the cornea, may later prove to be of value in the matter of prognosis of partial cataract, but at present their significance is not understood.

The Test of Refraction Method.

Retinoscopy, the use of the ophthalmometer and refractometer are all valuable aids in giving the oculist a lead as to the proper lens for the individual eye, but the final test for correct refraction in a case of cataract must be a subjective one. The patient must cooperate, especially in selecting the axis of the cylinder for the correction of astigmatism.

The test may be made either with or without a mydriatic, but for purposes of comparison at subsequent visits, the same method should be used in the same individual to avoid any discrepancy due to the 1. or 2. diopters of accommodation[4] remaining in eyes of patients of cataract age.

Of the 945 total cases of lens opacity observed, 241 were followed for an average period of 4.8 years.

Averages for All Types of Cases of Lens Opacity.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
241	92	149	69	206	35	447

The outstanding work of Vogt on the use of the slit lamp microscope has been constantly studied by the author. The excellent treatises on various subjects by Graves, Koeppe, Koby, Butler and Blaauw have also been used for reference.

Vision	Eyes	Average Change in Vision	Average Change in Refraction	Maximum Change in Refraction	
Improved	23	.15	+ .02	Hyperopic	+ 2.00
Same	226 (50.5%)	...	— .13	Myopic	—10.00
Diminished	198 (44.3%)	.30	—1.14	No change	36.5 %

Average Expectancy of Change of Refraction 63.5%

Averages serve as a great balance. The progress or arrest of all partial cataracts is not to be rated by them. On the other hand, it is the striking occasional case which is most apt to be remembered and to influence our judgment.

It may serve our purpose better to analyze the incidence of the various types of lens opacities and the influence of the progressive types on refraction.

Incidence of Various Types of Lens Opacities.

Congenital Cataract. Cataracts which had their origin in the prenatal period were observed in 62 patients, .78% of the total number of cases of lens opacity.

Incidence.

Cases	Male	Female	Both Eyes	One Eye	Total Eyes
58	36	22	34	24	92

Types.

Opaque Embryonic Sutures.....	5 Cases
Opaque Hyaloid Remains.....	22 Cases
Axial Embryonal Cataract.....	14 Cases
Embryonic Nuclear.....	6 Cases
Ectopia Lentis.....	6 Cases
Unclassified	5 Cases

The recognition of such a condition gives its significance immediately.

Illustrative Cases.

Case No. 1. C. S. Male, aet. 39. Vision each eye with glasses .9. The reduced central visual acuity due to macular changes. The left lens shows an opaque anterior "Y" suture, cerulean in color.

Case No. 2. M. K. Female, aet 30. Vision each eye with glasses 1. Remnants of the anterior vascular tunic of the lens in the form of gossamer strands attached to the region of the

lesser circle of the iris, and star shaped pigment cells on the anterior lens capsule. Cases of this type are not listed in the series, as pupillary remnants in some form are seen in almost every case.

Case No. 3. M. S. Female, aet. 28. Vision each eye with glasses 1. Just nasal to the posterior pole of the lens is a dense white opacity identified as nodular remains of the hyaloid artery. (Cataracta polaris posterior spuria.)

Case No. 4. C. B. P. Male, aet. 39. Vision each eye with glasses 1. Just posterior to the anterior "Y" shaped embryonic suture is a fluffy white opacity identified as axial embryonal cataract, developed from the cells which are left in the center of the lens vesicle and pushed forward by the development of the posterior cells of the vesicle into fibers.

Case No. 5. T. S., aet. 10. Vision O. D. with glass .6; O. S. with glasses .9. The ophthalmoscope shows a nuclear opacity, seen with the aid of the microscope to be composed of minute sand and colored opacities in the embryonic nucleus. (C. centralis pulverulenta.) No cases of Vogt's spear cataract or of anterior or posterior polar cataract with reduplications were seen in this series.

Zonular Cataract. Three cases of zonular cataract located in the zones between the embryonic nucleus and the capsules were seen in children. One was in a boy who had a history of rachitis.

Traumatic Cataract. In 47 patients, 4.97% of the total number of cases of lens opacities, traumatic cataract was observed. Of these, 22 were caused by contusions of the eye; 25 by perforating wounds. 33 were in male, 14 in females. All involved one eye.

It is beyond the scope of this thesis to describe traumatic cataract other than to bring out a few significant points by illustrative cases.

Case No. 1. G. N. Female, aet. 40. O. D. injured by contusion 1 year ago; O. S. destroyed at the same time by perforating wound. The right lens showed a thin layer of posterior subcapsular cortical opacity. There were a few fine pigment deposits on the posterior lens capsule, and fine macular changes. In the presence of a posterior cortical lens opacity, a history of even slight contusion of the eye may have significance.

Case No. 2. N. L. Female, aet. 31. Suffered a perforating

wound of the right eye by the point of a scissors when she was a child of four years. Adherent synechiae are evident with an anterior capsular opacity (the opacity is subcapsular, as the glass membrane of the capsule always remains transparent). A linear opacity and reduplications of the subcapsular opacity extend back thru the cortex to a zone in the infantile nucleus.

The latter case demonstrates that by the depth of the opacity one may, after sufficient cases are recorded, be able to estimate the time of the development of various lens opacities.

Coronary Cataract. Coronary lens changes of some type may be found in nearly all lenses. Vogt[5] has found the wreath type in 25% of all individuals beyond puberty. Most of these are to be seen only by direct illumination, due to their relucency, while they are invisible by retroillumination or the ophthalmoscopic test, as most of them are transparent. Vogt considers them a pathologic lens change. They do not however, by themselves lead to complete opacification of the lens. Coronary cataract may later be complicated by lamellar separation or water splitting of the sutures, and then progress to maturity.

In this series, only those cases which were visible with the ophthalmoscope, or which encroached upon the axial area so as to interfere with vision were included. 43 cases, 4.55% of the total were observed.

Incidence.

Cases	Male	Female	Both Eyes	Age of Greatest Incidence
43	11	32	43	50-55

Effect on Refraction.

27 patients were followed an average of 4.4 years.

Cases	Male	Female	Average Age	Both Eyes	Total Eyes Observed
27	8	19	50	27	54

Vision	Eyes	Average Change in Vision	Average Change in Refraction	Maximum Changes in Refraction	
Improved	0	0	0	Hyperopic +	.75D
Same	44	—	—.02D	Myopic —	.25D
Diminished	10	.38	+.15D	No change 55	%

ILLUSTRATIVE CASES.

CASE No. 1. M. W. Female, aet. 42. Showed numerous linear, punctate, round and club like opacities in the peripheral cortex. They were all of a uniformly brownish color on focal illumination. They tended to encroach upon the axial area, but did not interfere with vision. The homatropin acceptance was as follows:

May, 1922, O.D. —.87 Sph. ⌒—.12 C. ax. 165=1; O.S. —.25 Sph. ⌒—.25 C. ax. 165=1.

May, 1923, O.D. —.62 Sph. ⌒—.12 C. ax. 165=1; O.S. —.25 C. ax. 165=1.

June, 1927, O.D. —.50 Sph. ⌒—.25 C. ax. 165=1; O.S. —.37 C. ax. 165=1.

CASE No. 2. J. A. Female, aet. 43. Showed numerous coronary opacities, many of them being cerulean by focal illumination. Homatropin acceptance:

May, 1922, O.D. +1.00 C. ax. 90=1; O.S. +.62 Sph. ⌒+.50 C. ax. 90=1.

Feb., 1925, O.S. +1.00 C. ax. 90=1; O.S. +.50 Sph. ⌒+.50 C. ax. 90=1.

These cases showed practically no change in refraction or vision over a period of five years in the first case and three in the second.

CASE No. 3. Senile punctate opacities thru cortex. F. S. Female, aet 56:

May, 1923, O.D. —.50 Sph. ⌒—.75 C. ax. 10=1; O.S. —.37 Sph. —.75 C. ax. 20=1.

April, 1926, O.D. —.75 Sph. ⌒—.75 C. ax. 30=1; O.S. —.62 Sph. ⌒—.50 C. ax. 40=1.

Vision and appearance of punctate opacities remained the same.

STAR SHAPED CATARACT. This type of opacity of the central suture systems of the lens was observed in 9 cases, .95% of the total cases of lens opacity.

Incidence.

Cases	Male	Female	Both Eyes	One Eye	Total Eyes
9	4	5	3	6	12

Location of Star Shaped Opacity.

Embryonic nucleus 4; Infantile nucleus 3; Adult nucleus 2. This type of opacity is a nonprogressive one, as the factors concerned with its development are past.

Illustrative Cases.

Case No. 1. C. S. Female, aet. 84. Opacity in the posterior embryonic suture of the right eye. Vision with glasses .3. Nuclear sclerosis was also present. The opacity in the posterior embryonic suture was evidently developed during fetal life.

Case No. 2. G. J. E. Male, aet 48. The right eye shows a star shaped opacity, visible with the aid of the ophthalmoscope, and identified with the aid of the slit lamp microscope as being composed of iridescent points in the form of a seven point star, in an outer anterior and posterior zone of the adult nucleus. Etiology unknown. Vision with glasses O. D. = .9.

Cortical Striae. Opaque striae, radially arranged, may be found in the peripheral cortex. These may have their origin in water split fissures in which insoluble opaque salts have been deposited, but their frequency without any sign of intumescence would indicate that they may form simply by the deposition of opaque material along certain suture lines. They may remain stationary for years. They are found both in the anterior and posterior senile cortex, and symmetric areas may be involved, but inasmuch as the anterior and posterior suture lines never fuse, the anterior and posterior striae are not joined at the equator.

Incidence.

No. of Cases	Percentage of Total Opacities	Male	Female	Affecting: Both Eyes	Affecting: One Eye	Affecting: Total Eyes	Vision Reduced
75	7.94	30	45	48	27	123	38 eyes

Age of greatest incidence, 60-65.

Effect of Cortical Striae on Refraction.

18 cases of cortical striae were observed for an average period of 10 years.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
18	3	15	62	17	1	35

Vision	Eyes	Average change in vision	Average change in refraction	Maximum changes in refraction	
Improved	1	.2	+.25	Hyperopic	+ 1.00
Same	19	—	+.07	Myopic	— .25
Diminished	15	.25	+.16	No change	55%

Three other cases in combination with coronary opacities followed these averages.

ILLUSTRATIVE CASE.

M. E. K. Female, aet. 65 (1909). Peripheral striae noted. No evidence of intumescence.

June, 1909, O.D. +3.00 Sph.=.6; O.S. +3.50 Sph.⌒+2.00 C. ax. 180 = .5.

Nov., 1920, O.D. +2.75 Sph. ⌒+.50 C. ax. 20=.6; O. S. +3.50 Sph. ⌒+2.00 C. ax. 180=.6.

Mar., 1925, O.D. +3.00 Sph.⌒+.50 C. ax. 20; O.S. +3.75 Sph.⌒+2.00 C. ax. 180.

CORTICAL WEDGE SHAPED OPACITY. Thin flat layers of opacity may form in various symmetric lamellae of the senile cortex. The end of the wedge is blunt, and the anterior joined to the posterior at the equator. The axial end is sharp, rounded or zig-zag. These may be multiple in the same lamella or in different lamellae. They are found occasionally associated with lamellar separation. This fact suggests their origin. However, their frequency without any lamellar separation or other sign of intumescence would indicate that they may arise without intumescence. These cortical wedges were found in 33 cases, five of which showed also lamellar separation.

Incidence.

Cases	Percentage total opacities	Male	Female	Both Eyes	Affected One Eye	Total Eyes	Age of Greatest Incidence
33	3.49	15	18	11	22	44	55-60

EFFECT OF CORTICAL WEDGES ON REFRACTION.

11 cases were followed for an average period of 6.1 years.

Case	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
11	2	9	58	10	1	21

Vision	Eyes	Average change in vision	Average change in refraction	Maximum change in refraction	
Improved	0	0	0	Hyperopic	+2.00
Same	13	—	+.07 D	Myopic	—.50
Diminished	8	.12	+.43 D	No Change	43%

ILLUSTRATIVE CASES.

CASE No. 1. A.H.J. Female, aet. 65, 1925. Peripheral wedge found in left lens.

Nov., 1925, O.D. +1.00 Sph.⌒+.50 C. ax. 115=1; O.S. +1.00 Sph.⌒+1.00 C. ax. 85=1.

Mar., 1927, O.S. +1.25 Sph.⌒+.50 C. ax. 115=1; O. S. +1.25 Sph.⌒+.75 C. ax. 85=1.

Case No. 2. A.M.D. Female, aet. 48. Vision each eye with glasses .9. In the infranasal quadrant in each eye, in the mid zone of the senile cortex, was a thin anterior and posterior flat wedge shaped opacity, composed of fine dusty brown granules. Just anterior to this opacity the lamellae were separated.

Case No. 3. D.P. Male, aet. 50. Observed 5 years. No change.

Vacuoles. Vacuoles (globules or spherules) may be found in various layers of the senile cortex, often in or just beneath the epithelium of the lens. The contents of these vacuoles is usually clear fluid, but due to their spherical shape and difference in refractive index as compared with the surrounding tissue, they cast shadows back into the lens substance when viewed by direct illumination. Single vacuoles may often be detected best by using marginal retroillumination. Whether or not single or multiple vacuoles occurring without signs of lamellar separation or water splitting of sutures can be regarded as a sign of incipient intumescence of the senile cortex, is a matter for future research. The nature and significance of these isolated vacuoles in otherwise normal lenses is still a problem. They can be detected with the ophthalmoscope.

Vacuoles occur frequently in incipient, immature, intumescent and mature cataract, and in posterior cortical cataract. They seem to be part of the condition. In this series, only isolated vacuoles, occurring without any other sign of intumescence, are listed.

Incidence of Lens Vacuoles.

Number of Cases	Per cent of Total Opacities	Male	Female	Both Eyes	One Eye	Total Eyes	Age of greatest Incidence
173	18.31	75	98	55	118	228	45-50

They were accompanied by changes other than those of intumescence in 31 eyes, being found with nuclear sclerosis in 17, cortical striae in 6, cortical wedge in one, and coronary in 7 eyes.

Effect of Vacuoles on Refraction.

44 cases were followed an average period of 5.7 years.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
44	19	25	51	39	5	83

Vision	Eyes	Average Change in Vision	Average Change in refraction	Maximum Change in refraction	
Improved	2	.1	+.18	Hyperopic	+1.00
Same	70	—	+.10	Myopic	—.25
Diminished	11	.12	+.44	No Change	59%

Two cases in combination with peripheral striae followed these averages.

Illustrative Cases.

Case No. 1. M.F.W. Female, aet. 39. A single anterior subcapsular vacuole in the left eye. Homatropin acceptance:

Oct., 1923, O.D. +1.75 Sph. ⊃+.50 C. ax. 135=1; O.S. +.25 C. ax. 30=1.

Feb., 1926, O.S. +2.00 Sph. ⊃+.25 C. ax. 145=1; O.S. +.25 Sph. ⊃+.25 C. ax. 30=1.

Case No. 2. F.H.U. Female, aet. 28.

Jly., 1914, O.D. +.50 Sph. ⊃+.37 C. ax. 65=1; O.S. +.25 Sph. ⊃+.37 C. ax. 105=1.

Oct., 1925, O.D. +.75 Sph. ⊃+.25 C. ax. 75=1; O. S. +.75 Sph. ⊃+.25 C. ax. 105=1.

Developed a single vacuole in each eye. Feb. 1927. Acceptance, vision and vacuole remain unchanged.

Senile Cortical Cataract.

The manifestations of cataract in the senile cortex are those of hydration or intumescence. The water binding power of the lens as a whole has been changed thru some unknown factor and excess water is taken up. This produces disturbances in the normal arrangement of the lens fibers and sutures of the senile cortex.

Nuclear, posterior cortical, coronary, and other forms of cataract are often complicated by, and complete opacity of the lens produced by this process of hydration and disintegration of the senile cortex. After maturity, it is practically impossible to tell whether one of these types preceded the intumescence, except in the case of heavily sclerosed nuclear cataracts, where the amber colour of the depths of the lens will disclose this fact.

A total of 395 senile cortical cataracts of various stages were seen.

Total Cases	Percent of Total Opacities	Male	Female	Both Eyes	One Eye	Average Age
395	41.8	175	220	180	215	65-70

Incipient Stage. Lamellar Separation and Water Splitting of Sutures.

Lamellar separation and water splitting of sutures are the most common signs of incipient senile cortical cataract. They may occur alone, or together, or in combination with vacuoles. In classifying them, it is best to link them, as they are clinically all part of the same process, beginning hydration or intumescence of the senile cortex.

Lamellar separation is most frequently found in the inferior nasal area, but can involve any area or layer of the senile cortex, usually in symmetric layers anteriorly and posteriorly, and affects sometimes the outermost layers of the adult nucleus. It shows as almost parallel, thin, slightly irregular folds and depressions, producing fine alternating light and dark lines.

Water splitting of the suture lines of the senile cortex usually occurs from the equator toward the lens center. Infrequently the central suture may be separated, either in the same or in different layers of the senile cortex, both anterior and posterior.

The sutures are at first distended by clear fluid, but later there are globules which have clear fluid, but cause opacity by the difference in their index of refraction. Opaque material is later deposited in these globules and in the fissure, and an opaque spoke may be produced.

Incipient senile cortical cataract (lamina separation and water splitting alone or in combination) was observed in 168 cases. In 39 of these cases, it was combined with pathologic nuclear sclerosis, and in 19 with posterior cortical cataract. The senile cortical changes occurred alone in 110 of these cases.

Incidence of Incipient Stage.

Cases	Per cent of Total	Males	Females	Affecting Both Eyes	Affecting One Eye	Age of greatest incidence
110	11.64	38	72	69	41	70-75

Immature Stage.

Progress of the incipient cataract is made thru more widespread lamellar separation and water splitting and vacuolation of the cortex. Morgagnian globules appear in the split sutures, and opaque material is deposited between the separated

lamellae, along the sides of the split sutures, in and around the vacuoles. The fibers undergo a homogenous or granular degeneration, and the clinical stage of immature senile cortical cataract has been reached. The nucleus is frequently but not always involved.

Clinical classifications are useful but often artificial. In this series, immaturity was taken as the stage when the opacity in the lens had progressed so as to reduce the vision to .5 or less.

Incidence of Immature Stage.

Cases	Percent of Total Opacities	Male	Female	Both Eyes	One Eye	Age of greatest Incidence
54	5.72	23	31	12	42	65-70

Effect of Development of Incipient and Immature Stages of Senile Cortical Cataract on Refraction.

24 cases were followed over an average period of 5.7 years.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
24	9	15	67	19	5	43

Vision	Eyes	Average Change in vision	Average Change in refraction	Maximum Change in refraction	
Improved	4	.15	—.25	Hyperopia	+.50
Same	15	—	—.30	Myopic	—2.00
Diminished	24	.31	—.25	No change	39%

Illustrative Cases.

Case No. 1. Observed since 1898. J.B.S. Female, aet. 69 (1919). Homatropin accept.:

Feb., 1919, O.D. +1.75 Sph. ⊃+.25 C. ax. 105=1; O.S. +2.00 Sph. =1. Lenses clear.

Apr., 1923, O.D. +2.25 Sph. =.6; O.S. +2.25 Sph. =.6.

The patient was observed with the ophthalmoscope to have incipient cortical cataract. A slight hyperopic change was manifest, even tho the vision had diminished.

July, 1927, O.D. +2.50 Sph. =.6; O.D. +2.00 Sph. =.6.

The lens changes were identified with the slit lamp microscope as water split sutures partly filled with opaque globules. A slight hyperopic change was evident in the right eye with a slight myopic change in the left.

Case No. 2. F.M.S. Female, aet. 66, 1921. Observed since 1890.

Feb., 1921, O.D. +.75 Sph. ⊃+.25 C. ax. 165=1; O.S. +1.00 Sph. =1. Lenses clear.

May, 1925, O.U. Lamellar separation found in senile cortex in each lens.

Feb., 1927, O.D. +.75 Sph. ⊃+.25 C. ax. 180=.6; O.S. +.75 Sph. =.6.

Lamellar separation in this case produced but a slight myopic change in refraction in the left eye.

Case No. 3. J.S. Male, aet. 63, 1922. Observed since 1901.

Feb., 1921, O.D. +1.50 Sph. =1; O.S. +1.75 Sph. =1. Lenses clear.

Aug., 1926. O.D. +1.62 Sph. =.9; O.S. +1.75 Sph. =1.

Lamellar separation and multiple anterior subcapsular vacuoles were found in each lens. The development of these features did not change the refraction.

Intumescent Stage.

More and more water is being taken up, the lens swells and the stage of intumescent cataract is accomplished. There are many vacuoles, forming practically a subcapsular mat. One sees in the anterior cortex the widely split sutures filled with translucent material separating the bundles of fibers, which appear grey-white and glossy. Intumescence is greater in the younger, softer cataracts. The anterior chamber is usually shallow in this stage. Eleven cases were observed.

Cases	Percentage total Opacities	Male	Female	Both Eyes	One Eye	Total Eyes	Age of greatest incidence
11	.85	9	2	3	8	14	55-60

Mature Stage.

Complete loss of transparency is produced in the intumescent stage. After the complete disintegration of the fibers of the senile cortex has reduced them to a granular or pultaceous mass, the lens begins to lose water and to assume its former proportions. The glass membrane of the lens capsule always remains transparent. There are many anterior vacuoles in and beneath the epithelium. The cortex is greyish-white, with granular or irregular flaky or lumpy opaque material replacing all normal cortical structures. 123 cases were observed.

Cases	Percentage total	Male	Female	Both Eyes	One Eye	Total Eyes	Age of greatest Incidence
123	13.02	61	62	37	86	160	65-70

Hypermature Stage.

Further loss of water may occur, the lens shrinks, the capsule is wrinkled and appears in folds and depressions. Many vacuoles are still present. Dense white opaque material in irregular disposition is seen in the place of the cortex. 8 cases were observed.

Cases	Percentage Opacities total	Male	Female	Eyes Both	Eye One	Eyes Total	Age of Incidence greatest
8	.85	5	3	1	7	9	65-70

Morgagnian Stage.

By a further process of disintegration, the cortical material is reduced to a milky fluid, the capsule is flaccid, and the sclerosed nucleus sinks to the dependent part. Two cases were observed, one in a male patient and one in a female. In each, only one eye was so affected.

Posterior Cortical Cataract. Posterior cortical cataract usually begins with the appearance of vacuoles in the posterior subcapsular layer. Between and around these, irregular opaque material is deposited. The opacity spreads laterally, so that the tendency is for the formation of a thin subcapsular, saucer shaped opacity honeycombed by vacuoles. If the opacity is eccentric, it may at first not interfere much with vision, but if it is at the posterior pole of the lens, great reduction of vision will result, even tho the opacity be small.

Various other types of posterior cortical cataracts are seen. Vogt has described a type which tends to break down the posterior cortex in an irregular fashion and to extend axially forward. Vivid iridescence is seen. He considers this physical type of cataract to be complicated.

A beautiful rosette shaped posterior cortical cataract may be found after certain contusion injuries of the eye.

A peculiar type of cataract rapidly advancing, some cases of which show no other sign of complicated eye diseases, we have in this series classed under posterior cortical, as the opacity is at first chiefly posterior cortical. There appears also a thin sheet of vacuoles and opacity beneath the anterior capsule, in and beneath the epithelium. There may or may not be iridescence. Maturation may be brought about by intumescence of the senile cortex. This type was seen in 19 cases, 7 male and 12 female. In 14 cases, no evidence of complicating eye disease was observed, in 5 cases there was evi-

dence of iritis in one, chorioretinitis in one and uveitis in two, and one in a case showing nystagmus due to papillomacular atrophy. These known complicated cases might be a variation of the Vogt type of complicated cataract, but there was no tendency for the opacity to extend axially forward.

Incidence.

Posterior cortical cataract occurred alone or in combination with other types of cataract in 136 cases, 14.39% of the total number of opacities. It occurred alone in 89 cases.

Cases	Male	Female	Both Eyes	One Eye	Total Eyes	Age of greatest Incidence
89	43	46	38	51	127	50-55

Effect of Posterior Cortical Cataract on Refraction.

26 cases were followed for an average of 6.5 years.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
26	13	13	53	19	7	45

Vision	Eyes	Average Change in vision	Average Change in refraction	Maximum Change in refraction	
Improved	4	.15	+.06	Hyperopia	+1.00
Same	20	—	0	Myopic	—1.75
Diminished	21	.23	—.44	No change	44%

One case associated with coronary opacities followed these averages.

Illustrative Case.

Case No. 1. C.C. Female, aet. 49, was observed for 7 years, and on the last visit was found to have developed in the left eye a posterior cortical, honeycomb opacity just temporal to the axial area. The poor vision in the right eye is due to macular changes. Homatropin acceptance:

Dec., 1920, O.D. +1.00 Sph. ⊃+3.50 C. ax. 98=.2; O.S. +1.75 Sph. ⊃+.50 C. ax. 15=1.

Dec., 1923, O.D. Do.; O.S. +1.75 Sph. ⊃+.50 C. ax. 35=1.

July, 1927, O.D. Do.; O.S. +2.25 Sph. ⊃+.75 C. ax. 45=.9.

The development of this opacity had reduced the vision .1 and had produced +.50 D. change in refraction.

Case No. 2. W.A.F. Male, aet. 44, was observed to have in each eye opacities and vacuoles beneath the anterior and posterior capsules. The opacity was chiefly posterior, showed no iridescence and no tendency to extend forward in the irregular manner described by Vogt as indication of complicated cataract. There were a few pigmented deposits on the

endothelium. The aqueous was normal. Homatropin acceptance:

Apr., 1926, O.D. —.37 Sph. ⊃+.75 C. ax. 15=.5; O.S. —.75 Sph. ⊃+1.00 C. ax. 150=.5.

Oct., 1926, O.D. —2.00 Sph. =.1; O.S. —.75 Sph. ⊃+1.00 C. ax. 90=.6.

Jan., 1927, O.D. —3.50 Sph. =.1; O.S. —.50 Sph. ⊃—.75 C. ax. 90=.9.

The tendency for this type of cataract is to progress fairly rapidly, and to produce a myopic tendency as the vision diminished. In the left eye, as the vision improved the myopia diminished slightly.

Pathologic Nuclear Sclerosis. As the lens develops and new lamina are added during the entire life of the lens, the older lamina are crowned to the central area, and by a process of sclerosis form the nucleus of the lens. By the age of 45 years, the accommodation of the average lens has been reduced to such a degree that presbyopia has resulted. The process of physiologic sclerosis does not interfere with transparency.

Frequently, however, pathologic nuclear sclerosis is observed, usually involving to a greater degree certain zones of the nucleus. It is evident as an indefinite haze when viewed with the ophthalmoscope, while with the aid of the slit lamp microscope, one observes an increased relucency of the nuclear zones with a typical yellow or amber color. The sclerosis may progress and involve the inner layers of the senile cortex. In some cases, the entire lens seems to be pathologically sclerosed. The color may change to a mahogany or cherry tint. (Cataract nigra.) The color appears more dense posteriorly, due to the posterior cortex being viewed thru the superimposed anterior and nuclear layers. The anterior and posterior zones of the nucleus become sclerosed equally and simultaneously.

Incidence.

Pathologic nuclear sclerosis (nuclear cataract) may be found alone or in combination with other types of lens opacities. It occurred in 220 cases, 23.28% of the total number of opacities. It occurred alone in 114 cases, 12.07% of the total opacities.

Cases	Male	Female	Both Eyes	One Eye	Total Eyes	Age of greatest Incidence
114	45	69	103	11	217	70-75

It occurred in combination with peripheral cortical striae in 10 cases, with post. cortical opacities in 28 cases, with incipient and immature senile cortical cases in 68 cases.

Effect on Refraction.

40 cases unassociated with any other type of lens opacity were followed an average of 6 years.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
40	14	26	71	37	3	77

Vision	Eyes	Average Change in vision	Average Change in refraction	Maximum Change in refraction	
Improved	5	.14	+.15	Hyperopia	+.75
Same	23	—	—.76	Myopia	—10.00
Diminished	49	.32	—2.23	No change	17%

Three cases in combination with cortical striae and one with coronary followed these averages.

Illustrative Cases.

Case No. 1. N.P. Female, aet. 62, 1918. O.S. more marked nuclear haze than O.D.

Oct., 1918, O.D. —2.50 Sph. =.6; O.S. —2.50 Sph. =.3.

June, 1920, O.D. —3.50 Sph. =.6; O.S. —4.50 Sph. =.3.

June, 1927, O.D. —9.00 Sph. ⌒—.50 C. ax. 180=.3; O.S. —10.00 Sph. =.2.

Case No. 2. F.R.S. Female, aet. 76 (1920).

Aug., 1920, O.D. —.75 Sph. ⌒—1.00 C. ax. 155=.6; O.S. +.50 Sph. ⌒+.50 C. ax. 150=1; O.U. Central haze of lens observed.

Jan., 1924, O.D. —2.50 Sph. ⌒—.50 C. ax. 155=.4; O.S. —.50 Sph. ⌒+.75 C. ax. 135=.6.

Mar., 1927, O.D. —3.50 Sph. ⌒—.50 C. ax. 155=.2; O.S. —1.25 Sph. ⌒—.75 C. ax. 45=.5.

With the increasing pathologic sclerosis of the nucleus, the lenticular myopia is increased. The slit lamp microscope was used to rule out any other type of lens change.

Important Mixed Types of Cataract.

Posterior Cortical and Pathologic Nuclear Sclerosis. This type occurred in 28 cases, 2.96% of the total number of cases of lens opacities.

Incidence.

Cases	Male	Female	Both Eyes	One Eye	Total Eyes	Age of Greatest Incidence
28	11	17	17	11	45	70-75

Effect of Refraction.

8 of these cases were followed for an average period of 4 years.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
8	6	2	65	5	3	13

Vision	Eyes	Average change in vision	Average change in refraction	Maximum change in refraction	
Improved	0	0	0	Hyperopia	0
Same	3	—	—1.91	Myopia	—8.50
Diminished	10	.35	—2.40	No change	0

Illustrative Case.

W.R. Female, aet. 65, 1922. Had been myopic all her life.

Dec., 1922, O.D. —5.50 Sph. ⌒—.50 C. ax. 155=.9; O.S. —4.50 Sph. ⌒—.25 C. ax. 30=1.

July, 1927, Nuclear sclerosis with amber hue and a slightly eccentric posterior cortical opacity of vacuoles, and thin posterior subcapsular opacity had developed.

O.D. —14.00 Sph. =.3; O.S. —6.50 Sph. ⌒—.50 C. ax. 30=.6.

Lenticular myopia has developed with the development of the cataracts.

Posterior Cortical Combined With Incipient and Immature Senile Cortical Cataract. This type was observed in 19 cases, 2.01% of the total number of cases of lens opacity.

Incidence.

Cases	Male	Female	Both Eyes	One Eye	Age of Greatest Incidence
19	7	12	14	5	65-70

Sufficient cases were not followed to get a proper average of the effect on refraction.

Pathologic Nuclear Sclerosis Combined With Incipient and Immature Senile Cortival Cataract. 68 cases of this type, 7.19% of the total number of opacities, were observed.

Incidence.

Cases	Male	Female	Both Eyes	One Eye	Total Eyes	Age of Greatest Incidence
68	31	37	25	14	64	70-75

Effect on Refraction.

37 cases were followed an average of 6.4 years.

Cases	Male	Female	Average Age	Both Eyes	One Eye	Total Eyes
37	17	20	67	27	10	64

Vision	Eyes	Average change in vision	Average change in refraction	Maximum change in refraction	
Improved	6	.18	0	Hyperopia	+.50
Same	17	—	—.77	Myopic	—5.50
Diminished	41	.39	—1.92	No change	15%

Illustrative Case.

E.W. Female, aet. 69, 1908.

Oct., 1908, O.D. +2.25 Sph. ⊃+.75 C. ax. 180=.9; O.S. +2.50 Sph. ⊃+.50 C. ax. 180=.9.

Dec., 1923, O.D. +1.75 Sph. ⊃+.75 C. ax. 180=.7; O.S. +.50 Sph. ⊃+1.25 C. ax. 165=.7.

Cortical changes were observed in the right lens.

June, 1927, O.D. +1.00 Sph. ⊃+.75 C. ax. 180=.5; O.S. —1.00 Sph. ⊃+1.25 C. ax. 165=.4.

The cortical changes are identified as water split sutures with clear droplets in the fissures, located at various depths, both anteriorly and posteriorly, in the senile cortex. The nucleus is hazy and pathologically sclerosed. The tendency in this case is for the development of lenticular myopia.

Comment on the Effect of the Development of Lens Opacities on the Refraction.

Coronary cataracts, peripheral striae and wedges, vacuoles by themselves, on the average produce negligible changes in refraction. Posterior cortical cataract and incipient senile cortical cataract, as manifested by water splitting of the sutures, lamellar separation and vacuoles, produce on the average a low grade lenticular myopia. Nuclear sclerosis alone, or in combination with other lens changes, is most apt to produce the frequent and startling changes in the focus of the lens which are associated with the development of cataract. It is this which is responsible for the development of the higher grades of lenticular myopia, the familiar "second sight" of certain elderly people.

The lens in the lower animals is globular. The lens in the early human embryo is globular and simple sutured. As

the child develops into the adult person, the suturing becomes more complicated, the number of sutures increasing from 3 to about 9 main branches, with about 15 smaller branches. Year by year, as the lens continues to develop new fibers, the older fibers are compressed to the center of the lens, forming the lens nucleus, the normal sclerosis of which produces presbyopia. This is compensated for by the flattening of the cortex at the periphery, thus keeping the refraction in balance.

In pathologic sclerosis, the lenticular myopia is due to an increase in the index of refraction of the sclerosed nucleus. Duane[6] states this, but it is interesting to see his opinion borne out with the aid of the detailed observations made with the slit lamp microscope. Vogt has recorded 3 cases of lenses of double focus, the inner lens being the sclerosed nucleus, having a definitely shorter radius of curvature, accounting in these cases for the lenticular myopia.

The fact that the myopia of certain cataracts is due to pathologic nuclear sclerosis is borne out also by the effect of removal of these lenses on the refraction, as compared with the effect of removal of other types of cataract.

In 25 cases, we had a record of a satisfactory test of refraction some time before it was necessary to remove the lens. The comparison of this test was made with the test of refraction after healing of the cicatrix was complete.

Table of Results.

Type of Cataract	Number of Cases	Average Change in Refraction
Senile Cortical	11	+12.41 D.
Posterior Cortical	1	+13.25 D.
Anterior and Posterior Subcapsular	4	+14.87 D.
Nuclear	4	+17.91 D.
Mixed Nuclear and Cortical	5	+16.50 D.

The greater change in refraction after the removal of the nuclear and mixed nuclear and cortical types of cataract indicates that the myopia of these cases is lenticular, and that it is abolished when the lens is removed.

Average Expectancy of Change of Refraction in Cataract.

From the statistics of cases of different types of lens opacities showing no change in refraction, one may estimate the likelihood of change of refraction in these types.

Type	Number of Cases	Years Observed	No Change Refraction	Average Expectancy of Change
Coronary	27	4.4	55%	45%
Cortical Striae	18	10.	57%	43%
Cortical Wedge	11	6.1	43%	57%
Vacuoles	44	5.7	59%	41%
Senile Cortical	24	5.7	39%	61%
Post. Cortical	26	6.5	44%	56%
Nuclear	40	6.	17%	83%
Nuclear and Post. Cort	8	4.	0%	100%
Nuclear and Senile Cort	37	6.4	15%	85%

The types which are most likely to show changes in refraction are the nuclear or the combination of posterior cortical or senile cortical with the nuclear.

Complicated Cataract. Inasmuch as the progress of certain cataracts will depend on whether or not there is present a complicating eye disease, such as detachment of the retina, uveitis, glaucoma, etc., it would be well if we could recognize some particular physical type of lens opacity which could be decided upon as pathognomonic.

Vogt has described an involvement of the posterior subcapsular suture system, often resette shaped, but breaking down into an irregular porous structure composed of many vacuoles between the opaque material. There is a tendency to ignore the normal anatomic lens structure, and to extend axially forward into the posterior cortex. A vivid iridescence of the specular zone of the posterior polar area and often of the peripheral zones is evident. Anteriorly, there is a thinner but similar lesion. The opacity progresses rapidly. This series includes 8 such cases and they were all in eyes complicated by other disease.

Analysis of the total number of cases of lens opacity shows that 124, 13.4%, were in eyes known to be complicated by other disease; 62 of these patients were male, 62 were female. In 76, both eyes were affected, in 48 only one eye was affected. Various physical types of lens opacities occurred in various complicated eye conditions. No particular type was pathognomonic.

Summary and Conclusions.

While engaged in a clinical study of the possibility of the prevention or arrest of cataract, it was found necessary to

set up standards for judgment of results. It was essential to formulate a set of conditions under which two workers could independently make their observations on the same patient and arrive at uniform conclusions.

Four practical standards were considered: 1. The Test of Refraction. 2. The Test with the Ophthalmoscope. 3. The Test of Refraction and 4. The Test with the Slit Lamp Microscope. The various factors concerned in these tests must be standard, and identical conditions used in all reexaminations.

By a consideration of the changes of refraction in various cases in which the exact nature of the lens opacity was detailed with the aid of the slit lamp microscope, it was found, that in this series, pathologic nuclear sclerosis, occurring alone or in combination with other lens changes, is accountable for the frequent and startling changes of refraction which occur in the development of certain cataracts.

One is struck by the multiplicity of forms of cataract. This means that for an accurate prognosis, it will be necessary to consider each one individually. They do not have the same significance. Collective averages cannot be made to apply.

No particular physical type of cataract was found to be pathognomic of a coexisting complicated eye disease.

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DISCUSSION.

Dr. Arthur J. Bedell, Albany, N. Y.: Dr. Kirby's communication last year was much appreciated. In his paper today, he deals with the slit lamp findings in the various forms of cataract and draws a large table of comparative expectancy of cataract. He has, however, not used the most exact method of recording lens changes—photography. I have here a set of stereoscopic photographs of practically all types of cataract.

He has further stated that the progress of the opacity was based on changes which took place in the pupillary area. This is unfortunate. No method of recording should be considered which does not state the size of the pupil, and no report should be given without the dilatation of the pupil. We must view the entire lens. Further, if visual improvement or

deterioration is a method of determining lens changes, then we should have a dilated pupil for all tests and not use improved correcting glasses.

We must clearly separate the cases of simple cataract from those associated with other ocular changes. No table can be drawn for many years. All sections of the country and world must be studied before any chart can be properly made.

Dr. R. H. T. Mann, Texarkana, Tex.: I wish to speak of my clinical experience in the treatment of incipient cataract. For more than three years, I have been treating these cases with dionin, and have learned some things about its use: First, it does no good in patients where the vision is not 5/10 or better. The treatment is hopeless with poor vision. However, in patients who have good vision, better than 5/10, the cataracts have remained stationary, and there has been considerable improvement in vision in many of these cases. It will not cure every case nor will it benefit every case, but I certainly have seen no such number of cases with incipient cataract improve before this treatment was begun. The patients come in at intervals of a month to have the vision tested, and in many of these cases there is an improvement in the vision after the use of dionin. One patient particularly, a man whose duty it was to watch the insulation glasses on the railroad telegraph poles, came because he could not see to work. In a month or two, he was better and was at work. After three years, his vision improved from 6/10 to 8/10, which is his vision at the present time. He is still working, and yet he had a beginning senile cataract in each eye. I could report a dozen or more cases of this kind. I am sure, where we get these cases early, that there is an improvement in vision in many of them.

Dr. Allen Greenwood, Boston, Mass.: I want to call your attention to one thing in the paper and let you draw your own conclusions, which I think will be similar to mine.

Dr. Kirby writes that very few cases showed any improvement, altho the patients said they felt better and saw better. I think that all will agree, that the psychology of our efforts must always be taken into consideration, and that what the patient says he observes is no scientific criterion of what has been going on. We are all trying to help our patients and make them more comfortable, and if with the psychologic stimulus plus good hygiene and care of the metabolism we can make our patients more comfortable, I think a lot of good has been accomplished.

I hope this work of Dr. Kirby's may be continued and that it may be broadened. I trust it will take in some of the standards Dr. Bedell has suggested, and that various other methods that are common in cataract work will be investigated.

Dr. F. W. Dean, Council Bluffs, Iowa: In July, 1921, I began the treatment of senile cataract with injections of 20 minims of a 1 to 5000 solution of cyanid of mercury, injecting it deeply in one dose under the conjunctiva. At the Montreal meeting, I reported the result in 129 of my patients. Up to that time I used no treatment except the cyanid of mercury, as I wanted to see whether it would accomplish anything, and it did in a number of cases. Case 3, July 26, 1921, had a vision of 20/30 in the right eye and 20/50 in the left. He let me inject the left eye, but said he did not want me to spoil the right. It was a cataract that was coming on rapidly, and there were a number of cortical striae. I examined

all my cases with the ophthalmoscope and did not use the slit lamp at all. His vision the first month went to 20/30. October 5, 1921, the vision of left eye equaled 20/20-1. The vision of the right, untreated eye went to 20/40 from 20/30 by this date. He then let me give that eye a treatment. When next examined, February 11, 1922, vision of right eye equaled 20/20-3; vision of left eye, 20/20. At the present time patient has a little better than 20/30 vision in both eyes.

Another case was a man with beginning cataract in the right eye; there were a few marginal cortical striae, but vision equaled 20/20; the left eye had a vision of 20/70. That was also a cortical cataract. He was a railway engineer, and had been told he would have to get better glasses or they would take the engine away from him. I gave him this treatment Dec. 17, 1921. Feb. 7, 1922, the vision was 20/30 in left eye. The last time I examined him, about a year ago, he had vision of 20/20 in the right eye and 20/40 in the left eye. He is still running an engine. (I have corrected my reports of these two cases from the records since returning to my office.)

Since our Montreal meeting, in the Fall of 1924, in addition to using cyanid of mercury, I have been using dionin two days in the week and also turning the patient over to an internist for treatment.

It is the cases of cortical cataracts which improve. In nuclear cataracts, I do not get improvement unless the cataract is complicated by changes in the cortex. I do not inject a nuclear cataract any more unless I want to hold the one eye until the other is ready to be operated.

The size of the pupil of course has to do with nuclear cataract. If you dilate the pupil, the patient will see fairly well in the margins around the nuclei; but in cortical cataract, if you dilate the pupil, you get less vision. I believe I get some real benefit from this treatment and will continue it in cortical cataracts, where the markings are at the periphery and the pupil area is simply hazy, with vision not below 20/70.

Dr. Daniel B. Kirby, New York City, N. Y., (closing): I thank the gentlemen for their very kind discussion. Unfortunately, the brevity of time to which I was limited for the presentation did not permit a full consideration of all details which will appear in the printed paper.

As to the matter of noncomplicated cataracts as compared with complicated type—these cataracts which I spoke of were all in noncomplicated cases. The averages I spoke of apply only in the cases we considered. They would not apply to any other part of the country, and perhaps not to the series of another man. We considered only the cases in hand, to have some uniform standards for comparison with the cases which we treated.

Psychologically, this treatment by ionization was a success; but unfortunately, when judged by the standards we set up, we could not consider it a success.

A REPORT OF TREATMENT OF EIGHTY CASES OF CATARACT BY IONIZATION.

By Daniel B. Kirby, M.D.

NEW YORK CITY, NEW YORK.

Numerous remedies, both local and general, have been tried by various investigators in an effort to produce absorption or arrest of senile cataract. In our efforts to find some means of influencing the nutrition of the lens in the living body, it was decided to give ionization of the eye a trial.

There was evidence that: (1) the process of the formation of incipient senile cortical cataract is one of hydration or intumescence of the senile cortex, as is beautifully demonstrated with the aid of the slit lamp microscope, the first signs being lamellar separation and water splitting of the sutures. The salt content and the water binding power of the lens protein have evidently undergone a change. (2) The process of disintegration or death of tissue outside the body is accompanied by liberation of acid ions. It is possible that the beginning disintegration of the lens cortex in the formation of incipient senile cortical cataract may be accompanied by a local increase in the hydrogen ion concentration.

There was a bare possibility that ionization of the eye might favorably influence these factors. The prospects were that improvement, if it were to occur, would be evident within a short time. The method of treatment was one which would insure the continued interest of the patients, and would give the opportunity of following various types of cataract regularly over a period of time, so that observations might be made with the aid of the slit lamp microscope on the physical changes that occur in the lens during the progress of cataract.

Cantonnet, Mawas and others have tried ionization without success in certain cases of cataract. However, we decided to exhaust its possibilities.

Material was available from the outpatient department of the eye service of Dr. J. M. Wheeler at Bellevue Hospital.

Certain patients of 50 years and over were examined particularly for evidence of lens opacities. Of these, 100 were selected for treatment by ionization. They were not all cases of cortical cataract. Some were even complicated by conditions which were more serious than the presence of lens opacities. But it proved to be a series of favorable and unfavorable cases on which one might base a judgment of results.

Four of the cases had relucent features in the lens, as viewed with the aid of direct illumination, which were transparent when viewed by retroillumination. They were the 3 cases of senile punctate coronary dots, and one case of iridescence of the anterior lens capsule.

Material.

Cases Selected for Treatment.

Lens Opacity	Cases	Male	Female	Both Eyes	One Eye	Average Age
Incipient Senile Cortical....	30	16	14	18	12	63
Immature Senile Cortical...	11	7	4	8	3	64
Nuclear	10	3	7	9	1	63
Nuclear and Senile Cortical	3	2	1	0	3	63
Ant. and Post. Subcapsular.	10	4	6	5	5	51
Isolated Vacuoles.....	16	9	7	1	15	61
Coronary Senile Punctate...	3	0	3	3	0	59
Cortical Striae.....	4	1	3	1	3	58
Cortical Wedges.....	5	1	4	3	2	57
Star Shaped.....	3	2	1	0	3	62
Iridescent Points in Cortex.	2	0	2	1	1	54
Posterior Cortical.....	2	2	0	1	1	58
Iridescence of Ant. Capsule.	1	1	0	1	0	52
Total.....	100	48	52	51	49	59

Males, 48; Females, 52; Average age, 59; Both eyes affected, 51; One eye affected, 49.

Thirteen cases presented complicated conditions.

2 cases of retinitis and macular lesions.

1 case an old iridocyclitis (one acute exacerbation during the treatment).

2 cases healed interstitial keratitis.

1 case chronic glaucoma with optic nerve atrophy.

2 cases diabetes.

4 cases nephritis with vascular hypertension.

1 case lues, secondary optic nerve atrophy.

1 case old toxic amblyopia (alcohol).

The standards for judging of the progress or arrest of senile cataract which have been written of in another article are partly the outcome of this study. They were not all

formulated when the work was started, but it was soon seen how necessary it was to have standards. If a patient had a similar lens opacity in each eye, only one eye was treated while the other was held as a control. Visual tests were made with 28 foot candles of artificial illumination falling on standard Snellen test charts.

A special record was made of the test of vision, refraction, the ophthalmoscopic and slit lamp findings, before treatment was begun. The patients were reexamined and comparisons made after one month's treatment.

Technic of Treatment.

Three pieces of apparatus for the production of a graduated galvanic current for ionization of the eye were operated by an able technician. Of the 100 patients selected, 80 followed the treatment with interest. In 6, a cup electrode containing a solution of Sodium Iodid (1-400) was used over the eye, while in 74, gauze or sponge electrodes impregnated with various solutions were employed. The opposite pole was placed over the occiput.

Cases	Solution used and polarity
29	Sod. Iodid (1-400) Positive pole to eyes.
3	Sod. Chlorid (1-100) Positive pole to eyes.
9	Sod. Bicarb. (1-100) Positive pole to eyes.
15	Potass. Chlorid (1-100) Positive pole to eyes.
12	Sod. Chlorid (1-100) Negative pole to eyes.
12	Sod. Bicarb. (1-100) Negative pole to eyes.

29 patients were treated every day for a varying period, 51 every other day and 5 at irregular intervals.

11 patients received over 50 treatments, 9 from 40 to 50, 19 from 30 to 40, 33 from 20 to 30 and 8 from 10 to 20 treatments.

The treatments averaged 15-20 minutes. A current of 1-3 milliamperes was used with the cup electrode, while 2-6 milliamperes were used with the gauze or sponge electrodes.

The immediate reaction after the treatment was that of hyperemia of the skin and conjunctiva. Two of the cases treated with the cup electrode suffered corneal epithelial lesions, which healed promptly. A number of the patients complained of vertigo upon arising from the treatment chair.

Treatments were carried on from Feb. 15th to June 30th, while observations were continued thru July, 1927. Seven

patients were observed from 3 to 4 months, 21 from 2 to 3 months, 36 from 1 to 2 months and 16 less than one month.

Results.

21 patients expressed themselves as well pleased with the results in their cases, saying that they could see better. In 14 of these, however, this was not confirmed by test. Some of them could read another line with the treated eye, but they could also read better with the untreated eye. Four of the others could read an average of .1 better. The refraction had changed in these an average of +.43 D. The others were not given a final test. In two cases, anterior subcapsular vacuoles disappeared. In no case was any improvement seen in cases of lamina separation or water splitting of the fissures. Two cases of anterior and posterior subcapsular opacities showed progress by the development of more vacuoles and the lateral spread of the opacity; in one case, 2 Morgagnian globules in a water split fissure, which had been noted as "clean vacuoles in the fissure," became filled with greyish opaque material. Most of the cases exhibited no change in the opacities.

Conclusions.

Taken as a whole, the treatment of these cases of cataract by ionization must be ranked as a failure. The margin of improvement over untreated cases is not sufficient. In the matter of the disappearance of the two vacuoles, it must be said that 14 other cases of vacuoles were treated and showed no change. In no untreated case did vacuoles disappear.

The two manifestations of incipient senile cataract, lamellar separation and water splitting of sutures, which it was thought might be influenced by ionization, were not favorably affected in any case.

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TRANSACTIONS

OF THE

THIRTY-SECOND ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OTOLARYNGOLOGIC SESSIONS

ETIOLOGY, PATHOLOGY AND TREATMENT OF HYPERESTHETIC RHINITIS.

FRANK J. NOVAK, JR., M.D.

CHICAGO, ILLINOIS.

One may approach the problem of hyperesthetic rhinitis as a study of an allergic state. In that case, one is greatly concerned with what the patient is sensitive to, and the phenomena of sensitization are of paramount importance. On the other hand, if one cares less about what a patient is sensitive to and is more concerned with the question of why he is sensitive at all, using the word sensitive in the allergic sense, one must pause and ask the pertinent question, "What evidence is there to prove that all types of hyperesthetic rhinitis are forms of allergy?" Before proceeding any further there it may be well to define terms.

Vasomotor rhinitis, hyperesthetic rhinitis, hay fever, rose fever, hyperplastic ethmoiditis, hyperplastic ethmosphenoiditis, are terms which are sometimes used indiscriminately to label one of a group of pathologic conditions. If I may be permitted to classify these conditions by placing them into two groups, I shall call one group "Seasonal hyperesthetic rhinitis," and the other group "Nonseasonal hyperesthetic rhinitis." While this simple classification is not accurately descriptive, it serves to separate definitely those vasomotor conditions which are strictly seasonal in occurrance, and with which there is no concern in this paper, from the condition which is nonseasonal in character, and which I shall try to show is neither essentially allergic in character, nor is it a disease entity, but rather a symptom of a general systemic disturbance. By nonseasonal hyperesthetic rhinitis, I refer to th condition characterized by paroxysmal sneezing, and profuse, thin, watery discharge from the nose occuring at any time of the year. The sneezing occurs usually with its greatest violence in the morning upon arising.

It occurs regardless of exposure to irritating vapors, perfumes, dust, smoke, face powders, horse emanations, cat fur,

feathers, etc., etc. It seems, however, to be excited or increased in some cases by exposure to one or more of these agents. Young females are more prone to these attacks than the male. The condition may be aggravated during the hay fever season. It is described by many patients as a continual cold in the head. They are sensitive to changes in the temperature of the surrounding air. They suffer a great deal from the cold in the winter. They avoid drafts, they shun bathing in cool or cold water. They feel comfortable in the superheated atmosphere of the modern steam heated flat. Rhinoscopy reveals a pale boggy mucosa. The pallor, which is always marked, may be limited to the mucosa of the middle turbinate and to the adjacent area of the nasal septum. It may involve the lining of the entire nasal cavity, however. The soft tissues of the interior nose are waterlogged, soft, boggy, shiny in appearance, and are covered by a thin, watery secretion. While the patients in advanced cases may be sensitive to one or many things, and will go into a paroxysm of sneezing upon the introduction of a soft cotton application into the nose, they require but a minimum of cocainization for surgical manipulation without pain. The histopathology of the turbinate, and other nasal tissues according to Beck, is as follows:

The mucous membrane is fairly well preserved. The subepithelial tissue is edematous. The bone is invariably rarefied, with large marrow spaces. Sometimes one of these marrow spaces is so distended as to form a bony, turbina bullosa. The lining of this bulla is endothelial; the fluid contained is of mucin like character. The above applies also to the ethmoid labryinth.

These patients run a consistently subnormal temperature, averaging about one degree below normal. There is a slight arterial hypotension. This observation is made on the basis of blood pressure readings on about one-hundred cases of nonseasonal hyperesthetic rhinitis. The pulse is usually slow. The pulse was taken with the patients seated, and after a fifteen minute rest. The average pulse in the series was sixty-eight (68), the slowest being forty-eight (48) and the most rapid (in a heart case) being ninety (90).

Most of these patients have a dry skin, rather coarse, dry and brittle hair, and nails. They are easily fatigued by bodily exercise, and prefer muscular rest to muscular activity.

It was the preceding observations recurring persistently which forced the question, whether or not the local condition, this sneezing and discharge, is but a symptom of a general underlying condition. The natural line of inquiry was regarding the blood chemistry in these cases. The most consistently recurring finding was a hypocalcemia, and in the small series observed up to the time of 1924, this was a most persistent finding, and I was led, prematurely to be sure, to the conclusion that hypocalcemia was the basis of the disease. Since that time, I have found, in accordance with others, that a low calcium content of the blood serum is by no means a constant finding in nonseasonal hyperesthetic rhinitis, and I wish to go on record at this time to that effect. I believed until 1924, that thyroid extract tended in some way to help increase the calcium content of the blood serum. At that time, I started studying the basal metabolism of every patient who would submit to the test, regardless of the patient's complaint. It soon became evident that every patient who had a nonseasonal hyperesthetic rhinitis also had a low basal metabolic rate, and moreover, that the severity of the symptoms was in direct proportion to the degree of hypothyroidism. The following case illustrates this fact:

The patient is a woman of fifty. She is overweight, soft, flabby, blonde. For the past six years, she has suffered from frequent attacks of sneezing (paroxysms of forty or more sneezes), and the discharge of copious amounts of thin, watery secretion from the nose. She underwent a partial bilateral middle turbinectomy and ethmoid currettage four years ago.

EXAMINATION.

Ears: Normal.

Nose—Right:

1. Evidence of turbinate operation.
2. Mucosa pink, except in region of middle meatus where it is pale and boggy.
3. Clear, thin, water secretion in nose.
4. No polyps, no pus.
5. Septum straight, no spurs, etc.
6. Inferior turbinate boggy.

Nose—Left:

1. Evidence of turbinate operation.
2. Mucosa of entire nasal chamber very pale.

3. Thin, watery secretion in nose.
4. No polyps or pus.
5. Septum normal, but pale.
6. Inferior turbinate is hypertrophied.

Pharynx: Normal except for dilated capillaries on post-pharyngeal wall. Tonsils atrophied.

Larynx: Mucosa pale.

GENERAL: Pulse (Average) 72. Temperature 98.8° F.

Her temperature was generally subnormal, but during one period of five days, during a "cold," the average temperature was brought to normal by a temporary hyperpyrexia.

COMMENT.

In this case the basal metabolism was low. On certain occasions the rate was as low as minus thirty. Upon the administration of thyroid extract, grains one-fourth, three to four times per day, the metabolic rate was increased, and paralleling the increase in the rate of metabolism, a subsidence of the symptoms was noted. When the basal metabolism reached normal, the symptoms, sneezing, watery discharge, and mental depression disappeared. At this point, thyroid medication was stopped. After the lapse of three or four months, the symptoms returned and the basal metabolic rate was again found to be low. Thyroid medication was resumed, and the symptoms once more disappeared when the basal metabolism reached a point within normal limits. This required a period of eight to ten days, and the patient was again free from her symptoms. She has been under observation for over two years, and it is necessary to keep her metabolism up by the periodic administration of thyroid extract, in order to keep her free of symptoms. This case is typical and shows in a striking manner the dependence of the patient's well being upon the rate of her basal metabolism. It should be noted in this case, as well as in the others, that there were times during the period of two or three years when the administration of thyroid extract failed to improve the symptoms, but on investigation it was found that the thyroid extract had been purchased at a corner drug store, where it had lain on the shelves for a long time and had become inert.

The following is a tabulated list of twenty-five cases of nonseasonal hyperesthetic rhinitis, showing the low metabolic rate, and the simultaneous effect of thyroid medication on the symptoms and on the metabolism.

Name	Age	Sex	Symptoms	Duration	Aver. Temp.	Aver. Pulse	B.M.R.* Before Treatment	B.M.R.* After Treatment	Results
V. B.	34	F	Sneezing Discharge	8 mo.	99.2	84	—12	0	Cessation of Symptoms
Mrs. E. L. H.	30	F	Sneezing Discharge	2 yrs.	97.6	80	—3	0	Cessation of Symptoms
Mrs. C. T. L.	50	F	Sneezing Discharge	4 yrs.	97.6	72	—22	—4	Cessation of Symptoms
G. E. M.	30	M	Sneezing Discharge	1 yr.	96.8	56	—20	—3	Slightly Improved
Mrs. O. M.	40	F	Sneezing Discharge	2 yrs.	98.2	76	—23	0	Cured
R. N.	4½	M	Sneezing Discharge	2 yrs.	Sub-normal	Irreg.	Not done	Not done	Cured
Miss A. O.	21	F	Sneezing Discharge	3 yrs.	98.6	72	—12	0	Cured
Mrs. F. P.	35	F	Sneezing Discharge	....	98	64	—7	Plus 20	Cured
Miss B. J. R.	30	F	Sneezing Discharge	2 yrs.	99	84	—7	Variable	Much Improved

Name	Age	Sex	Symptoms	Duration	Aver. Temp.	Aver. Pulse	B.M.R.* Before Treatment	B.M.R. After Treatment	Results
Mrs. J. J. R.	30	F	Sneezing Discharge	1 yr.	98.8	96	—9	0	Cured
Mrs. R. P. W.	40	F	Sneezing Discharge	Long Time	98.4	60	—11	0	Cured
Mrs. O. B. N.	28	F	Sneezing Discharge	6 mo.	98.6	86	Plus 5	Plus 10	Cessation of Symptoms
Mrs. N. S. H.	38	F	Sneezing Discharge	3 yrs.	98	70	—12	0	Cessation of Symptoms
Dr. R.	40	M	Sneezing Discharge	10 yrs.	No record	..	..	..	Symptoms Disappeared
Mrs. I. P.	50	F	Sneezing Discharge	3 yrs.	98.6	72	—1	Plus 5	Entirely Relieved
Miss M. T.	24	F	Sneezing Discharge	2 yrs.	98	68	—18	—3	Entirely Relieved
Miss M. F.	19	F	Sneezing Discharge	1 yr.	97.6	78	—3	Plus 2	Cured
†Mrs. R. W.	25	F	Sneezing Discharge	10 mo.	98.6	78	0	0	Symptoms Disappeared

*B. M. R. abbreviation for basal matabolic rate.

†The basal metabolism test on Mrs. R. W. was not done under basal conditions. This was not known to us at the time of the test.

Name	Age	Sex	Symptoms	Duration	Aver. Temp.	Aver. Pulse	B.M.R.* Before Treatment	B.M.R.* After Treatment	Results
J. S.	8	M	Cretinism; Hyperesthetic Rhinitis	..	..	..	..	..	Myxedematous condition much improved. H. Rhinitis cured.
V. A.	6	F	Cretinism; Hyperesthetic Rhinitis	..	..	..	..	..	Myxedematous condition improved. Hyperesthetic Rhinitis cured.

*B. M. R. abbreviation for basal metabolic rate.

NOTE: The treatment of all these cases consisted of the administration of thyroid extract. (Hynson, Westcott & Dunning.)

In the beginning of this paper, I made a tentative classification of seasonal hyperesthetic rhinitis and of nonseasonal hyperesthetic rhinitis. Let it be said parenthetically, the seasonal type is undoubtedly an allergic disease, in which there is no hypothyroid basis and in which a hypocalcemia is rarely found. The nonseasonal type is probably nonallergic in character basically, but is a symptom of a myxedema, mild in character, in which the rhinoscopic picture reveals mucous membrane changes which are similar to those which are observed in more pronounced types of myxedema in the skin, hair, and nails.

Osler writing on myxedema says, "The swelling of the mucosa of the nose is a frequent source of irritation," and quoting further, he says, "Cleveland has called attention to swellings of the turbinate bones, of waxy pallor, improved by thyroid feeding." Curtis, as early as 1894, called attention to the possibility of the nasal mucosa being the first seat of the peculiar gelatinous infiltration of myxedema. Berard states, that in myxedematous subjects, the most habitual lesions at autopsy consist of infiltrations of the tegument and the mucosa by mucin; in thyroid myxedema, the cutaneous dystrophia and edema is not accompanied by intellectual defieciency, while in parathyroid myxedema it is. McCarrison, in the histologic examinations of a section of myxedematous skin, also observes that the skin and mucous membrane were

infiltrated by a substance resembling mucin, and that the mucous membrane of the nose was similarly affected. Lortat-Jacob and De Gennes state, that in myxedema, the endocrine disturbance apparently determines the appearance of skin and mucosal lesions, which resist all treatments except opotherapy. Leopold Levi and Rothschild say, that patients with hypothyroidism are especialy affected by the least disturbance of external temperature (coryza). The mucous membranes are pale, opaque, may be swollen like the skin, which is most obvious in the mouth and nose.

Treatment.

In treating a case of nonseasonal hyperesthetic rhinitis, it is essential to keep a careful record of the pulse, temperature and basal metabolism rate of the patient. To do this efficiently, the patient is taught to take the pulse at stated periods during the day, and to record it on a card furnished for that purpose. The same holds for the temperature. Before commencing treatment, a basal metabolism test is done. If that is within normal limits or is higher than normal, the futility of administering thyroid, not to say danger, is evident, and yet I had the pleasure of learning of a colleague of mine who prescribed thyroid extract for a patient without so much as taking the pulse, temperature, or determining the basal metabolic rate. This particular patient had an inflammatory rhinologic condition; she had a very rapid pulse; she had a high metabolic rate. She soon exhibited definite signs of a marked hyperthyroidism under thyroid medication. The inflammatory condition of the nose failed to improve, and my friend expressed the profound opinion, that he always had thought that this thyroid treatment of hyperesthetic rhinitis was all the bunk, and that now he was sure of it. This patient did not have a nonseasonal hyperesthetic rhinitis. She had a rapid pulse, and elevation of temperature, and a high metabolic rate, and this gentleman should have blamed his carelessness, his misinformation, and his stupidity instead of blaming the method of treatment.

The basal metabolism test, despite the declarations of manufacturers of apparatus, is not an easy test to do. It is not easy, because it calls for a meticulous regard for detail. To send a patient to a commercial laboratory, not knowing who the technician may be who will do the work, and to be

satisfied with one reading, is a fatuous disregard of what is necessary. The test must be done by a skilled and conscientious technician. It must be repeated, and the treatment must be guided by subsequent tests. For the fact is, that there is a perfect paralellism between the nasal metabolism rate of the hyperesthetic patient, and the degree of severity of his symptoms.

I have used small doses of Hynson, Westcott & Dunning Thyroid Extract, starting with a quarter of a grain, three times a day, the dosage always controlled by frequent basal metabolic tests. In those cases in which there is also a hypocalcemia, five grains of calcium lactate are given, three times a day, and the patient is advised to have general bodily irradiations with mercury vapor quartz lamp.

Conclusions.

(1). Seasonal hyperesthetic rhinitis, namely, hay fever, rose fever, etc., is probably a purely allergic condition.

(2). Nonseasonal hyperesthetic rhinitis is probably a symptom of an otherwise clinically unrecognized myxedema, and is not allergic in character.

(3). All cases of true nonseasonal hyperesthetic rhinitis have a low basal metabolic rate. The more severe the symptoms, the lower the rate, and vice versa.

(4). The administration of an active, fresh preparation of thyroid extract is logical treatment.

(5). This treatment must be guided by the results of frequent basal metabolic tests (done properly).

(6). In some of these cases, there is a hypocalcemia. The administration of the chlorid or lactate of calcium is indicated.

PROTEIN SENSITIZATION TESTS IN THE DIAGNOSIS of HYPERESTHETIC RHINITIS.

SIEGFRIED MAURER, M.D.

CHICAGO, ILLINOIS.

Hyperesthetic rhinitis is a new name applied to a group of symptoms, a part of which have been handed down to us along with the faith and folk lore of centuries ago. Eighteen centuries ago, it was considered an ill omen, and a blessing was bestowed upon the sneezer as a deprecation of the evil likely to arise. Other names bestowed upon this syndrome are coryza vasomotoris, vasomotor rhinitis, perennial rhinitis, and perennial hayfever. Hayfever is restricted to the seasonal symptoms. This condition is supposed to be associated with an inflammation of the nasal mucosa in which the nerves are hyperesthetic.

Its symptoms—sneezing, nasal discharge, and blocking of the nares—are normal physiologic acts accomplished for the protection of the organism against deleterious elements entering the respiratory tract.

The efferent nerves controlling this act take origin from the respiratory center. It is a complex coordinated act, in which one or two deep inspirations are taken and followed by a forceful expiratory effort, during which the glottis is widely opened and the mouth shut off from the nasopharynx. In this manner, the entire respiratory blast escapes thru the nares at high pressure, expelling mucus not only from that organ, but also from the throat. At the same time, other reflexes, such as increased secretion, are usually set up.

The distribution of the afferent nerves which initiate this complex coordinated act is not wholly understood. The optic, olfactory, auditory, and fifth cranial nerves are known to produce the symptoms of hyperesthetic rhinitis when they are strongly stimulated. The nerves involved when tactile and sensorial impressions produce these symptoms, are less understood. However, it has been observed often, that a sudden change of temperature of the surface of the body, or tactile

stimuli about the nose, may induce sneezing. The repetition of this reflex, with blocking of the nares and nasal discharge (the latter two to a greater or less degree), constitutes the syndrome of hyperesthetic rhinitis.

Abnormal strong stimulation of the optic, olfactory, fifth, or auditory nerves, produces the syndrome by reflex. Sudden bright light may stimulate the optic, loud noises the auditory and irritating substances the nasal division of the fifth, to initiate the reflex. Irritating substances may be gases that are inhaled, such as sulphur dioxid; or they may arise from an infection of the mucosa. The common cold is a good example of this type of infection. Patients with the foregoing etiology rarely seek the advice of a physician, because the cause is quite evident.

In many cases, however, the syndrome of hyperesthetic rhinitis cannot be traced by the patient to any obvious stimulus. In such cases, the nasal mucosa is found to be the seat of periodic swellings, which arise and subside much as do urticarial wheals of the skin. I believe that these periodic swellings have an origin identical with that of the wheals of urticaria.

Recent discoveries have made it possible to produce passive urticarial wheals, thus enabling us to analyze a condition that resembles the turgescence of the nasal mucosa. Prausnitz and Kustner showed that the skin of a normal nonhypersensitive individual can be passively sensitized by the intracutaneous injection of the serum of a person markedly hypersensitive to fish. The area so sensitized will produce an urticarial wheal upon the application, by intracutaneous injection, of a solution of the fish to which the donor was hypersensitive.

Following the experiment of Prausnitz and Kustner, Walzer and Walzer discovered that a wheal is obtained at the site of local serum injection when the recipient eats fish. A hypersensitive skin area is not produced if the subject eats fish and is simultaneously injected with the serum of a fish hypersensitive individual.

Hooker has shown that some individuals develop a reactivity of the skin upon the subcutaneous or intravenous administration of a small quantity of horse serum. Park, in earlier unpublished results, confirms Hooker's observation of a change from negative to strong positive reaction of the skin.

Park has given several of these individuals second intravenous injections of horse serum, without having any of them suffer the slightest constitutional reaction.

These experiments show quite clearly that the activating substance is liberated in the alimentary tract, is absorbed, and circulates in the blood.

Prausnitz and Kustner showed, that a person hypersensitive to a substance or group of substances can be made hypersensitive to a new one by the intradermal or subcutaneous injection of the new substance. This is not true of a person who has never suffered, or whose blood relations have never suffered, from any type of hypersensitiveness (eczema, hives, angioneurotic edema, asthma, hayfever, hyperesthetic rhinitis, or certain types of cough).

Certain individuals have hypersensitive mucous membranes. In such persons, simple contact of the irritating substance with the membrane will lead to the immediate swelling of the membrane. Cow's milk will, in certain infants, produce a profound swelling of the mouth the first time that the milk is fed. One of my patients, who is hypersensitive to nuts, suffers from a swollen mouth, a burning sensation in the mouth, throat, esophagus and stomach, followed by vomiting and a severe diarrhea, when she eats nuts. The swelling and vomiting may persist for 24 hours. From this we may conclude that the entire gastrointestional tract is hypersensitive in certain individuals.

The tissue of an individual that is afflicted with the hypersensitive idiosyncracy is different from normal tissue in a number of ways. When such tissue comes in contact with a suitable irritating substance, the cells produce something that is capable of specifically activating even normal tissue to the irritating substance. Some, or possibly all, of this activating substance is thrown out into the blood stream, and may be demonstrated in the serum by biologic tests. The activating tissue will then react with the irritating substance in such a way that swelling of the tissue occurs.

Tissue from a hypersensitive patient regenerates more of the active substance every time it comes in contact with the irritating substance, providing that activating and irritating substances are not present at the same time. Normal tissue that has been sensitized by bringing it in contact with serum that contains the activating substance, reacts only once, be-

cause it is incapable of generating the activating substance. The activating substance is destroyed when the tissue reacts with the irritating substance.

When the hypersensitive cells are contained in a non-absorbing membrane, such as the skin, the activating substance may be brought to it via the blood stream and tissue fluids, or by breaks in the continuity of the protecting surface.

A comparatively small hypersensitive area may produce activating substance, which finds its way into the blood stream, to be removed by other cells, sensitizing them. The secondarily sensitized cells react when the absorbed irritating substance has accumulated above the threshold of reaction.

As will be shown later, there seem to be two distinct kinds of hypersensitive individuals. In the one kind, the cutaneous tissue seems to be essential in the desensitizing process. In the other kind, the cutaneous tissue appears to have very little to do with desensitization, and in such patients it is the subcutaneous tissue that is directly responsible for desensitization. The subcutaneous tissue is involved in desensitization in all cases. I will also show later, that wheal formation on the mucous membranes of hypersensitive individuals may be of two kinds. In one case, the wheal is limited to a swelling of the cutaneous tissue. In such cases, the swelling rapidly appears and disappears at intervals. In other cases, the swelling is distinctly one of the subcutaneous mucous tissue, and in such cases the swelling persists for many hours or days.

Much has been said about irritating substances. What are they? No one knows exactly. Duke believes that mechanical and physical agents are capable of producing this type of reaction. Certain drugs, such as quinin, arsenic, and aspirin, seem in some cases to function as irritating substances. Of all substances used, protein containing substances, or something associated with them, seem most universally to act in this way. Most of the careful experimental work has been carried out with proteins or protein containing substances.

The literature contains several series of cases of this syndrome giving positive skin reactions to proteins. Piness' series of 130 cases shows 23.4% hypersensitive to pollens, and 76% to food, animal hair alone, or food and animal hair, or in combination with pollen. In this series, he gives examples

of two relieved by the removal of the offending protein. Those hypersensitive to pollens were treated by the injection of the extracts of the pollen. They would have obtained complete relief had they been removed from the pollen.

Eggston's series was of 150 cases of perennial hay fever (hypersensitive rhinitis). In 10% of these cases, the chief cause of the symptoms was protein sensitization. This figure is probably too small, for he did not subject his patients to complete tests.

Kahn reports two cases of perennial hayfever which were relieved of symptoms by the removal of feathers, wool and pollens.

Rich reports five cases giving positive reactions to orris root alone, all of which obtained relief when they abstained from the use of orris root face powder.

Floystrum and Krag report a patient who developed hyperesthetic rhinitis and hives on the exposed parts when unpacking hardware in straw. Extracts of the straw gave a pronounced local reaction on the skin. A cure was obtained by refraining from handling the straw.

Novak studied the metabolism of hyperesthetic rhinitis patients. He finds that there is a group which have lowered basal rate and low blood calcium. Blood calcium studies have been made exhaustively during the past few years. It has been found that hypocalcemia is indicative of no special type of disease. There are many patients who have low blood calcium, who do not have any of the symptoms of hyperesthetic rhinitis. The decrease in basal metabolism has been definitely proven to be related to a hypofunction of the thyroid gland. Some patients with hypothyroidism may also be so unfortunate as to have hyperesthetic rhinitis, but all patients with lowered basal rate do not have hyperesthetic rhinitis. A person with hyperesthetic rhinitis may have any other disease in addition, and the findings associated with the other diseases, such as hypocalcemia and lowered basal rate, are in no way diagnostic of the hyperesthetic rhinitis.

When one omits all of the seasonal type of cases, 10% of the remainder, according to Eggston, are due to protein sensitization. This is probably too low, but as yet we do not have a large enough series to set a more accurate figure.

How can one test for hypersensitiveness? It is essential that the irritating substance be brought in contact with the

hypersensitive cells. This can be done in two ways: (1) By intradermal injection of a solution of the substance you wish to test; egg, extract of orris root, etc. (2) By rubbing the dry powdered protein containing material into shallow incisions, over which a drop of O.IN sodium hydroxid has been placed.

The latter is the method of choice, because of the ease of making and reading the tests. The proteins are stable in dry form, so that the preparation of the sterile solution is dispensed with. Our method of application is as follows: The skin of the back is cleansed with dilute alcohol to remove all traces of protein derived from the clothing or dust. From eight to twenty-four shallow incisions are made, about one and one-half to two inches apart, in rows of two, four, or six, according to the size of the back. The best instrument for this purpose is the fresh broken edge of a thin razor blade held firmly in a small hemostat. These incisions should be made just deep enough to reach the top of the corionic papillae. A slight oozing of serum tinged with blood will follow. To each incision is added one drop of O.IN sodium hydroxid. A small amount of the powdered protein is then added, each incision acting as a test for one protein. Two incisions serve as controls. To insure that the protein goes into solution, and that the solution comes in contact with the freshly cut cells of the incision, each drop is rubbed with the smooth end of a small glass stirring rod. The rubbing is controlled so that each incision receives the same amount of rubbing and the same pressure. A blank is rubbed first and last. Reactions are read in 20 to 30 minutes after the blanks appear normal. Reading of the reaction constitutes drawing the size of the scratch and wheal in black, and the erethema in red. After the complete test is finished, one studies the size of the reaction and decides what is to be considered a definite positive, a doubtful and a negative. Socalled four plus positive reaction sizes are found to differ, depending on the power of the skin to react. What constitutes a four plus reaction in one person may be a doubtful reaction in another. The back has the advantage of being a large area to test, and it is also an area which has been protected, so that there is a better chance of it reacting. I have found skins of forearms that would not react to any protein, while the back showed a large reactions. Some area where the skin is subject to constant

pressure and trauma will not give reactions to either this technic or to intradermal injections of strong protein solutions.

The tests having been made, we must complete the diagnosis by producing relief from the symptoms by treatment. In some cases this is comparatively simple, and may consist in a change of diet, removing the foods to which the patient is hypersensitive, change of clothing, or a change of environment. When such changes are impossible, or when they fail to yield satisfactory results, desensitization must be resorted to, and this will necessarily be one of two kinds. Specific desensitization is used when feasible; that is, patients who are specifically hypersensitive to a limited number of substances in their environment, are injected with extracts that have been prepared from these substances. When specific desensitization is not feasible, we have to resort to nonspecific desensitization. There are many nonspecific desensitization agents, and they are injected either intravenously, intracutaneously, or subcutaneously. Salvarsan, neosalvarsan, neutral acriflavin, and sodium thiosulphate, are the ones most commonly used by the intravenous route. Of these, five to ten cubic centimeters of a 20% solution of sodium thiosulphate is probably the safest and most reliable. Peptone has been used intravenously, subcutaneously, intramuscularly, and intradermally. The intraveous application is apt to lead to a peptone shock, which is very discomforting to the patient, not to mention the physician. I have obtained very satisfactory results by injecting 0.05 c.c. of a 30% peptone solution subcutaneously, or by giving 0.1 to 0.3 c.c. intradermally. I use the intravenous method very little. The choice between the intracutaneous and subcutaneous methods is largely a matter of experiment, and some of my observations in this field may be of value.

The first injection is usually given intradermally, and must be made with not over 0.05 c.c. of solution. The concentration and age of the extract determines the size of the wheal, so that a weak solution is always injected first. Two distinct types of reaction are observed: (1) With a comparatively weak solution, a wheal is obtained promptly which grows rapidly in size by extension of its edges and by pseudopod formation. An area of erythema and sometimes itching preceeds the advance of the wheal. Later, the wheal begins to lose its white appearance, and its height is increased by a swelling of

the subcutaneous tissue beneath it. Should the dose be properly gauged, the symptoms will soon disappear or become less severe, depending upon the amount of irritating substance coming in contact with the reacting substance. Depending upon the state of hypersensitization of the patient, the relief may persist only part of the time the area is visibly reacting, or may continue after it appears normal. When the symptoms recur, the patient should have another intradermal injection, the size of dose being governed by experience.

(2) After an intradermal injection, which requires a relatively strong concentration of the irritating substance, the wheal grows rapidly for a few minutes, as did the first. It soon disappears. There is no swelling of the subcutaneous tissue. Very little relief is experienced from one of these injections. I have given as many as six such injections at one time to obtain some relief for the patient. In this type of patient, relief can be obtained by injecting the extract subcutaneously. The amount of relief depends upon the size of the dose. Too large a dose will cause an increase in the severity of the symptoms, or some type of general reaction, such as hives, asthma, marked toxic feeling with great swelling at the site of injection, which may include the whole extremity. Such a reaction is not infrequently associated with an elevation of body temperature, which may reach 105 degrees F. This reaction may last four or five days. Hot or cold packs applied to the swelling may intensify the systemic reaction. Repeated injections of adrenalin, in doses as large as can be tolerated, give the best relief. The injection of the irritating substance is repeated as often as symptoms return, or at intervals to forestall the return of the symptoms, until the course is completed. Hypersensitiveness to horse dander may be removed by subcutaneous injections of the extract of horse dander, at semiweekly intervals over a period of eight to nine months. This usually gives relief for about four years.

Failure to recognize the existence of these types of cases has led to considerable controversy and some distress. A patient belonging to class one will suffer from dangerous augmentation of the symptoms if he is given a subcutaneous injection of the irritating substance. An improper intracutaneous injection, either given too deep or in too large an amount of fluid, will be equivalent to a subcutaneous injection

and will lead to severe symptoms. It is usually advisable to test each person by giving an intradermal injection.

From the foregoing conclusions, it should be clear that hyperesthetic rhinitis is a disease that may originate in a large number of ways. One form of treatment that cures all cases is hardly possible.

We have, first, all those unusual cases that have their origin in a powerful sensory stimulation, such as a strong light, a draft of cold air, tactile stimulation about the nose and mouth. Then there are the cases of chronic, acute and subacute infection, and these are usually cured by clearing up the infection, which may necessitate an operation to correct anatomic abnormalities. And finally, there are the unfortunate individuals who have an inherited hypersensitive idiosyncracy. The outstanding characteristic of these hypersensitive individuals is their liability to exhibit abnormal cutaneous reaction, such as wheal formation, eczema, etc., whenever their tissue cells are brought in contact with irritating substances to which they are hypersensitive. The swelling of the nasal mucosa is merely a manifestation of this kind. Cutaneous urticarial wheals itch and burn. Wheals in the nasal mucous membrane will also itch and burn. The sensory stimulation so incited may excite the sneeze reflex, or the glands may thus be incited to secrete. The outcome will be the complete syndrome of hyperesthetic rhinitis.

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DISCUSSION OF DR. NOVAK'S AND OF DR. MAURER'S PAPERS.

DR. JOSEPH C. BECK, Chicago, Ill.: Anyone who has any kind of practice in ear, nose and throat diseases, will have had sufficient experience with the type of disease that Dr. Novak has described, which in times past has been a much overoperated condition. But in this presentation of Dr. Novak, it was a great relief not to find a word regarding surgery, operation on the ethmoids, the cautery, etc. Considering it from a much wider standpoint, that it is a local manifestation of a general disease, he was sure (and I am not) that it would be a great satisfaction, if he could go home now and feel that myxedema, and the symptoms subsequent to hypofunction of the thyroid, are responsible. It seems to me, if he wanted to prove that in another way, he could go to a clinic where there are a lot of thyroids overoperated, not by any fault, but simply a condition where they remove the mass and produce, for a time at least, myxedematous symptoms, and see whether these people really show the same condition in the nose as the case he referred to, which was a waterlogged nose.

There is an additional pathology which he did not mention, and that is that many of these cases are hyperplastic to the extent of polyposis and polypoid degeneration, and in such cases, surely treatment of any kind, even raising the metabolic rate, would not do any good.

As to the actual fact of raising the metabolic rate, we were much interested in this, and had the technician who makes these tests take a sufficient number of cases and test them to see, and sure enough there were a few with a low metabolic rate—not very low—but how many of these patients when they are at ease, with no excitement, would not show many times a fairly low metabolic rate? I am not willing to subscribe to that test as indicating a cause for this condition, but I do believe that this disturbance is a local manifestation of a general condition, and that it is a nutritional disturbance, that the people who have this trouble such as clerks, working in-doors, usually receive systemic benefit from quartz therapy. They do not eat proper food, usually selecting that which is devoid of fat, or they carefully remove it, and we do have these cases which do splendidly when we give them fats, especially cod liver oil. There is something wrong with their nutrition, and that has to do with the disturbed metabolism.

There is a great deal yet to be done in this work. Patients are not all free from infection. If the nose has been treated properly, they will probably get better, but the trouble is operations are still being done in these cases.

While I am supposed to discuss Dr. Novak's paper, I am much interested in the second paper, and I would like to ask the essayist if he has come across the recent work done by Stürm von Lueewen in study of allergies, particularly allergies like the molds, the aspergillus especially, and if he found these allergies were specific for asthma and could not be destroyed unless they were frozen to a tremendous degree—sufficient to destroy the colloid substance, which is a sensitive object? If he has, I would like to have his views on the subject.

Dr. W. V. Mullin, Cleveland, Ohio: I think these are valuable papers, but yet it seems to me we are as far from the solution as we were before.

Dr. Novak made one statement that was more or less in accord with what I said in discussing Dr. Duke's paper before this Academy last year, and that is that if we were more prone to investigate and find out why these people have a sensitivity in the first place, we would be much farther along.

Dr. Novak, I have enjoyed your paper very much. I think you have placed it now where it will do no harm. As long as you check your cases as often as you do by a metabolic rate, and as long as everything else is carried out as you suggest, I think you are entirely safe. I think, however, especially here in the Great Lake district, that if that rule is not observed and thyroid extract given, it will do a tremendous amount of harm.

Dr. Beck mentioned that cases that had symptoms of hypothyroidism might be observed to see if they had hyperesthetic rhinitis. I have had the opportunity to examine a number of cases after thyroid operations but have not noticed any nasal symptoms.

Dr. Beck said the condition might be nutritional and so it might, but it may also be racial.

As to the skin tests, I do not place as much value on them as the second essayist would give. We can rely on a great number of tests, and yet without a careful history and an environmental survey, they will not afford good results. The value of the skin test must be classified as to the protein that is looked for. Pollen and dermals are more satisfactory than tests for food. I believe, that in many individuals, there is a systemic reaction from the foods they take that will not show in any type of skin test.

In regard to the pathology in the nasal cavity, it is strange the different course these cases will follow with apparently the same irritant. Some remain hyperesthetic rhinitis all the way thru; others, as Dr. Beck has said, develop a hyperplastic rhinitis and polyposis and asthma. I have examined all the tissue that I have removed from these polypoid cases for a long period. The nasal cavity may be blocked with polypoid tissue, and yet that individual will not have, say bronchial asthma, nor will the tissue examined show eosinophiles. But the next patient, with the same amount of underlying sinus disease and polyposis, may, on examination of the tissue, prove to be loaded with eosinophiles, and the submucosal layers of the nasal tissues will be loaded with eosinophiles, and that individual will have asthma. I have thought that that was somewhat of a clue as to why some of these people have asthma and some do not. But we are not able to tell why they have eosinophilia.

Dr. A. H. Andrews, Chicago, Ill.: To some of us who are older in the profession, it has been very interesting to watch the progress of the papers which have been read before this society. A paper of twenty-five years ago would be materially different from those of today. The papers of today show progress. I do not think they are final, but they are making progress.

As long as I have been a teacher, I have been in the habit of saying to my students that hyperesthetic rhinitis is set upon a tripod, of which

one leg is a constitutional disturbance, another local pathology of the case, and third the external irritant. Among the external irritants are the pollens, foods, powders, etc. It is barely possible that I am mistaken about the second leg of this tripod; it may be it is not necessary to have local pathology in the nose; but with that exception, I think that gross classification will stand.

What the local pathology is, we are still in the dark. It has been recognized for many years that foods produce these symptoms. I knew one women who would be 98 if now living, who suffered from hyperesthetic rhinitis and urticaria whenever she ate certain articles of food; she also had asthma, which would return after taking these articles of food. Whether the trouble is, as Dr. Beck says, nutritional, or whether it may be a lowered metabolism, or something else we have not yet suspected, is food for thought and subject for investigation.

I am glad to see you left loopholes thru which we can crawl out. It is much safer than to make positive statements. I have made positive statements that I had a good deal of trouble in getting away from later, and my friend, Dr. Beck and I, have found it necessary to take back a good many things we have said, some of them in the meetings of this society.

But the great question before us is, What is the cause, and why? I am greatly pleased with Dr. Novak's report of his further investigations, and with Dr. Maurer's work; but as I said, they are not final. There is yet much to be done, and I am glad to see the younger men at work investigating these problems, for the great question in medicine that comes to us ever and over again, is why, why?

Dr. Robert Sonnenschein, Chicago, Ill.: I would like to congratulate Dr. Novak on the fairness of his statements. In some instances, he says he was mistaken, and I think he should be given credit. It takes a courageous man to say that.

In 1925, a paper was published in the Journal of the American Medical Association on the subject of hyperesthetic rhinitis, in which it was said that the blood calcium is invariably reduced in this condition. Dr. Pearlman and I were struck by this, but the laboratories to which we sent patients did not find the blood calcium below the normal rate.

As far as the basal metabolism is concerned, I feel with Dr. Maurer, that in cases of hypothyroidism you can have hyperesthetic rhinitis, but the fact that it is present does not say that it is the cause of the hypothyroidism. Dr. Simpson published a paper a few years ago, in which he showed that in a vast majority of cases, the hyperesthetic rhinitis did not show a lowered basal metabolism.

This excellent paper of Dr. Maurer, I believe, deserves our sincere commendation. The subject that he has discussed is a rather comprehensive one, of a highly specialized nature, and one to which only in comparatively recent years much attention has been paid. Whether these cases have become much more frequent of late, or whether our attention is called to them more definitely, it is hard to say, but I am inclined to think that both factors are present. The greatly increased use of cosmetics of various kinds has, I believe, contributed largely to the frequency of these cases, as shown by the fact, in our experience at least, that it is among young women that it is more often found. Orris root is present

in so many of the face powders, and this is one of the most common causes of the very annoying and distressing symptoms of the complex known as hyperesthetic rhinitis. Other facts of the etiology have already been mentioned by the essayist so that we cannot detail them in this brief discussion.

It is interesting to note that Dr. Maurer calls attention to the difference in the reactivity of the different parts of the body; for instance, the fact that the back is more sensitive because it is normally more protected. He says "some areas where the skin is subject to constant pressure and trauma will not give reaction to either this technic or the intradermal injection of strong protein solutions."

The diagnosis of the condition itself is easy as compared with the determination of the actual etiologic factors, which is often very difficult. We have noted, that while the protein sensitization tests in a very large number of cases have told us exactly what the cause of the symptoms is, we have often been disappointed in not being able to know after a most extensive series of tests just what is causing the patient's distress. In some instances, 80 to 100 different proteins have been tried, the patients found negative to all, and we were unable to find the exact cause of the symptoms. In our own practice, we have not done these tests but have referred the patients to a reliable laboratory, or to physicians paying especial attention to this work.

It has been our experience, that the calcium content of the blood is very rarely diminished below the normal range of from nine to twelve milligrams per hundred c.c. of blood. We have found likewise that the basal metabolism is seldom reduced in these cases.

So far as the therapy is concerned, we have had good results in many cases with the most simple measure, and in others the most detailed lines of treatment have availed very little, if any. One quite noticeable fact with reference to therapy is the number of agents recommended for the cure of this condition. In diseases such as syphilis and malaria, we have certain definite and specific agents upon which we can rely, and the number of drugs used is very small. When a long list of therapeutic agents is recommended for any one ailment, one may be quite sure that none of them is very efficient; otherwise the profession would soon learn to rely upon one or the other drug or procedure.

The medicaments which have given the most relief in our cases have been epinephrin preparations in dilutions of one to 16,000 or one to 8,000, or a two to three per cent ephedrin solution. Concentrated trichloracetic acid may be applied to the middle meati after thoro cocainization, and relieves many of the cases. Calcium chlorid applied to the nasal mucous membrane, used intravenously, or given by mouth, either as chlorid or lactate, will help many cases. Calcium, which acts as a powerful depressant, usually gives only temporary results, which disappear shortly after the drug is discontinued. So far as we are aware, ultraviolet radiation has been of benefit in only a very small number of cases; in most of them the results were disappointing, even when the radiation was applied by men of considerable experience. The rational therapy, it seems to us, is the elimination from the diet and environment of the patient of the

proteins to which he or she is sensitive, providing these can be found, or the desensitization of the patient to the guilty substance.

The whole subject of hyperesthetic rhinitis will require further intensive study for some time, before measures will be found which will to a desirable degree relieve this distressing and at times very obstinate condition.

DR. FRANK NOVAK, JR., Chicago, Ill. (closing): I expected to receive more violent treatment than I did.

Dr. Beck made one statement that I think is of great importance—something to the effect that many patients who have a hypothyroidism do not have hyperesthetic rhinitis.

I had an opportunity of studying only four true myxedematous subjects. I do not believe the hypothyroidism, which is temporary and not of great duration, can be called a myxedema. We have myxedema only when we have the symptoms of myxedema—symptoms in the skin, hair and nails—along with the other symptoms of hypothyroidism. When we have a hypothyroid subject who has not the pathology of myxedema in the nose, I do not believe we will get a hyperesthetic rhinitis. All four of the cases I studied at one time or another had definite hypothyroid symptoms. Carlson, in his work on hypothyroidism in dogs, made the observation that all of these dogs exhibited nasal symptoms; there was a thin, watery discharge from the nose, and they had a tendency to snuffle and sneeze.

The question of polyposis, I believe, is an end project; it is a finished state. A case of polyposis after hyperesthetic rhinitis is an extreme case.

Dr. Sonnenschein said one of his cases showed a low metabolic rate. All those I have studied have shown a low metabolic rate. There is a distinct parallelism between the rate and the clinical picture.

Dr. Mullin mentioned that hypothyroids are not all myxedematous or hyperesthetic. I think the same thing applies to other diseases.

Dr. Andrews said our findings are not final, and that is true. Perhaps my paper sounded dogmatic and gave the impression that we have reached a conclusion. That is not the case, and I should probably have used "probably" and "perhaps" a little more than I did.

DR. SIEGFRIED MAURER, Chicago, Ill. (closing): The higher bacteria mentioned by Dr. Beck are hard to kill by freezing. Some of them grow very well at freezing temperatures. The temperatures reached in our climate will not kill their spores. They have been used as allergens and give positive reactions with some patients. They are not specific for the symptom asthma, but may cause any of the allergic symptoms. They no doubt are one of the elements that make dust of certain localities obnoxious to some patients.

Dr. Mullin is dubious about placing much value on the skin test. I tried to make the point that the etiology of hyperesthetic rhinitis is manifold. In this day of medical specialization, patients seek the advice of a rhinologist. I am not a rhinologist, hence this group of patients is referred to me by the nose and throat specialists. The patients referred to me in this way are just the ones in which the skin tests are of value. Of the patients that are not referred to me, some might have been benefitted by skin tests and others as surely would not have been benefitted.

It is impossible, at this time, to judge accurately the relative values of different types of diagnosis and treatment or, indeed, to draw conclusions as to the relative prevalence of some one type of hyperesthetic rhinitis, because the current publications usually consider only some one type to the exclusion of others. Results that are of value can be obtained only by collection of a large number of cases by a group of competent diagnosticians.

I see no reason why Dr. Andrews should lose faith in the importance of local pathology. The local pathology may not be gross or microscopic in appearance, but on considering pathology from a broader sense, there certainly must be some type of local pathologic physiology. We have come closer to the "Why" of this pathology since we have learned to transfer the reaction from the nasal mucosa to the skin by proper intradermal injections. This transfer is accompanied by no particular discomfort.

I believe that Dr. Sonnenschein is in error when he considers a test out with 80-100 proteins to be adequate. Certain proteins are, of course, more important than others, but we are not in a position, today, to judge accurately the relative values of a large number of proteins. I do not consider a test to have been exhaustive unless it has included 400 proteins. There are cases, however, where a test with 80 proteins would be more than sufficient to locate the irritating substance.

TEN TRACHEOTOMIES AND THEIR LESSONS.

LYMAN RICHARDS, M.D.

BOSTON, MASSACHUSETTS.

The operation of tracheotomy, tho often a spectacular emergency, is not infrequently an elective procedure in the treatment of laryngeal obstruction. As such it takes its place with other less drastic measures, such as diagnostic laryngoscopy, bronchoscopy, or intubation, together with the well known nonoperative and palliative means of relieving this always grave condition. The decision as to which of these steps is indicated, the length to which they can be safely carried, and the degree of warrantable delay in their execution constitute a most important laryngologic problem.

During the past two years, at the Children's Hospital in Boston, we have been called upon to decide as to the advisability of tracheotomy in a group of children suffering from laryngeal obstruction of varying etiology. It is the purpose of this paper to present an account of this work, illustrated by concise examples, in which the operation of tracheotomy was performed with time enough for a preoperative consideration, and in some instances a trial, of other measures before final opening of the trachea.

First may be mentioned those cases in which prolonged rest of the larynx is the prime requisite. In prepapillomatous or papillomatous obstruction, it is certainly desirable to perform tracheotomy as a preliminary measure, since it not only provides a free airway in impending obstruction, but greatly facilitates the necessary after treatment of the larynx itself. If the degree of obstruction is relatively slight, it is a great temptation to delay opening of the trachea, and to begin local treatment without such precaution, but in our experience this has proved a mistake. In any such extensive operation as the removal of a true laryngeal tumor with the actual cautery, the resultant postoperative reaction will almost surely require a tracheotomy, perhaps under emergency circumstances, rendering it far more difficult and hazardous.

The indications for tracheotomy are less clear in the presence of laryngeal obstruction complicated by infection, and under these circumstances, decision as to operation has often proved difficult. In several instances, relief of the obstruction by opening the trachea has failed entirely to check the infection, which has extended downward to the bronchi and lungs with a fatal termination. The question arises as to whether the open tracheal wound has any tendency to produce this result or to contribute towards it, and hence whether at least preliminary palliative measures or intubation would not be preferable, a contention strongly held by some pediatricians.

Illustration of this point is the case of an Italian boy of six, who had been becoming more and more dyspneic. There was no history of foreign body, and his physician had treated him as a case of diphtheria. Laryngoscopy showed a narrow funnel shaped larynx, with cords so close together, that a small bronchoscope could not be introduced. In spite of the classical picture of laryngeal obstruction, the fear of doing a needless tracheotomy induced us to try palliative measures. Here, perhaps, intubation would have sufficed until the laryngeal swelling subsided, tho with such a narrow glottis, the risk of pressure and ulceration was considerable. There was no abatement of the symptoms, and tracheotomy was performed, revealing a very thick mucosa. There was immediate relief, and convalescence and decanulation were uneventful. In this instance, the operation was withheld as long as it safely could be, but eventually was necessary. The infection, however, was not nearly so acute as that seen in the younger children, where it tends to spread so rapidly to the bronchi and lungs.

In the following and more severe form, it presents a problem in therapeutics which calls for vigilance and persistence as is required in few other laryngologic conditions.

A six months old child was admitted for treatment of acute laryngeal obstruction and the familiar signs of increasing dyspnea. With the above case in mind, an attempt was first made to introduce an intubation tube, with the hope that the acute process would subside and an airway be maintained in the meantime. It was not possible, however, without the use of undue force, to insert the tube. For the next 48 hours, all the known methods of palliative treatment were tried at the suggestion of the pediatric department, who felt that trache-

otomy should be avoided if possible, and that the fatalities in some previous cases of acute streptococcus laryngotracheobronchitis might be due to secondary infection thru the tracheal wound. Stimulated by previous failures, we were determined to save this child's life if it could possibly be done. In 48 hours there was an increase in the dyspnea, and every one agreed that tracheotomy was the only proper procedure. Following this, the child was subjected to the most painstaking and energetic special nursing. He was breast fed by his mother, frequent suction was maintained in the tube and trachea, he was given antistreptococcus serum, a transfusion and whiskey. Moist saline was applied to the tube, expectorants given, and no effort spared at least to prevent the formation of the dreaded thick, tenacious crusts and secretions. In this we were successful, as the tracheal mucus was always thin and easily removed and never once caused any obstruction. At times the respiration rate went as high as 90, with a temperature up to 107° and a pulse rate of 176. Yet following this, there was a period, beginning 17 days after admission, when for five days the temperature was normal and we all thought we had won the battle. Within a few days, however, the temperature rose slowly but steadily, signs of consolidation appeared in the right lower lobe, and in 48 hours the boy was dead.

I have described this case in some detail, because it is representative of a difficult problem with which we are confronted each winter, that of treating this severe infection successfully. In each instance, the presenting symptom for which the patients seek relief is laryngeal obstruction. Faucial or laryngeal membrane is practically never seen, and the etiology must be attributed to a streptococcus infection. The outstanding point is the fact that complete relief of the obstruction in the larynx has only a temporary effect, and there next follows a severe toxic tracheobronchitis, which apparently terminates in a septic pneumonia. Our mortality had been disappointingly high, and reports from other men indicate a like situation.

When there is added to this picture of infectious laryngitis the possibility of a foreign body, a further difficulty is introduced. If, as the history may suggest, a foreign body is present, its removal is indicated before considering tracheotomy, but if not, then valuable time may be lost or further

damage done to the larynx, in searching for it. Occasionally, infectious membrane may simulate a foreign body and confuse the picture, as in the following instance.

A ten months' old baby, supposed to have picked up and inhaled some sort of nut shell, soon developed acute respiratory distress, with substernal retraction and cyanosis. Laryngoscopy revealed an edema of the arytenoids and a grayish-white object apparently lying between the cords. Efforts to grasp this were unsuccessful, but in some way it became dislodged or pushed downward out of sight, following which all signs of dyspnea disappeared and the breathing became essentially normal. In retrospect, the next procedure should have been a bronchoscopy to follow up the object in question, but in view of the marked improvement in the child's condition, nothing further was done. All went well until 12 hours later, when the same signs of laryngeal obstruction reappeared, and the question of the proper procedure was again raised. Thinking it over on paper, another laryngoscopy and probably bronchoscopy would be the logical method of tracing the cause of this reappearing obstruction, which certainly had the characteristics of some sort of foreign body. However, the previous unsuccessful laryngoscopy and the urgent condition of the patient led us to a tracheotomy, which at once relieved all the dyspnea. No sign of anything of a foreign body nature was seen in the trachea or coughed out. A laryngoscopy the next day showed a swollen larynx, no foreign body such as appeared at the first examination, but patches of exudate below the narrow glottic lumen. Culture for diphtheria was negative, but antitoxin was nevertheless administered. It was impossible to pass a 4 mm. bronchoscope thru the glottis.

Respirations and temperature continued to rise. Physical examination suggested a right bronchial obstruction. Since there were no crusts or inspissated secretions in the trachea, bronchoscopy thru the wound, tho seriously considered, was not done. Death finally ensued, and a postmortem report by the medical examiner stated only that there was no foreign body found.

Any death raises the question as to what might have been done to prevent it. Assuming there was no foreign body, in spite of the history of possible aspiration, bronochoscopy at the first examination would not have helped. The sudden

onset of the symptoms the next morning certainly suggested something loose in the trachea, and bronchoscopy then might have been of value, tho the larynx was soon afterward too tight to permit of this procedure. Intubation would not have permitted the possible escape of the supposed foreign body. Under these circumstances, in the face of serious dyspnea, tracheotomy seemed the wisest procedure.

That such situations may unexpectedly eventuate in tragedy, is evidenced by the story of an infant suspected of having aspirated a piece of popcorn and brought to the hospital with typical signs of increasing laryngeal obstruction.

The larynx, when viewed, showed redness and some edema, but permitted the easy introduction of a small bronchoscope, which failed to reveal any foreign body. A few moments after the completion of the operation, the dyspnea became so urgent that a tracheotomy was performed. The child was of the short necked, stout type, in which great difficulty was encountered in getting down to the trachea. Almost as soon as the latter was opened, there appeared, to the dismay of the operator, a subcutaneous emphysema below the chin. In spite of free breathing thru the tube and relief of the obstruction, this emphysema gradually spread until it extended down over the chest and abdomen into the legs. The fatal termination of this case, assuming it to be due to some fault in technic, has led us to perform all tracheotomies in a special position, in which, with a large sand bag under the shoulders, not under the neck, the head is held by an assistant almost in his lap in hyperextension, similar to the socalled Rose position in tonsillectomy. In this way, the trachea is brought forward close under the skin and is under tension, so that it can be readily palpated and located at any moment. The claim that this position gives rise to sudden laryngeal edema we have not found warranted. In this particular instance, it would have been better to reintroduce the bronchoscope and perform the tracheotomy more leisurely and over a surer guide. Here again, temporary intubation might have been a better procedure. The exact cause of the emphysema has never been quite clear, altho in some way a peritracheal break must have allowed air to escape the normal passage and find its way into the tissues. We have twice feared a similar occurrence after simple laryngeal dilation in the presence of tracheotomy wound, and found the slight emphysema of the neck to be

due to having the tapes too tight, whereby a sort of constriction was produced which trapped expired air as it passed the wound edges around the tube, until it appeared palpable under the skin. This disappeared immediately on loosening the tracheotomy tapes.

Laryngeal obstruction due to operative trauma has been another indication for several of our tracheotomies. A carefully performed bronchoscopy, not unduly prolonged and with proper sized tubes, does not usually lead to any postoperative edema, but in spite of all possible care, this condition does arise and must be handled promptly and efficiently. It is in this class of case, uncomplicated by infection, that we have come more and more to feel that intubation may have a valuable place, even tho our own experiences with it have not been particularly encouraging. If this procedure proves satisfactory, it is certainly to be preferred to opening the trachea. Our difficulty with intubation has been, that once the tube was in place and the initial obstruction satisfactorily relieved, it recurred again on removal of the tube. This necessitated reintroduction of the tube, presumably producing more laryngeal irritation and edema, and leading to a sort of vicious circle, to the point of making it impossible to remove the tube permanently. Exemplifying this is the history of a boy of 18 months of age, suspected of having aspirated a peanut, who was admitted for relief of wheezing and coughing. A first laryngoscopy showed so much swelling that it was impossible to pass a bronchoscope safely. Two weeks, marked by great diminution in symptoms, passed, during which the only positive evidence of foreign body was an X-ray report which denoted a general cloudiness of the left lung. A second bronochoscopy was nevertheless done, and a fragment of peanut located in the left main bronchus. A portion of this was removed, and the rest allowed to remain, it being thought better to remove it at a second sitting than to prolong the operation unduly. Shortly thereafter, there appeared all the signs of a loose tracheal foreign body, with intermittent obstruction at the glottis as the peanut fragment struck up against the cords. A second bronchoscopy was immediately done, and the remainder of the peanut removed without difficulty. Instead of the expected uneventful convalescence, there soon appeared a typical laryngeal obstruction, for which the only

explanation was the added trauma of the second operation, making us wish that it had been completed at one sitting.

In the light of other experiences, we thought that intubation might possibly tide this patient over the purely reactionary inflammation of the larynx, and thus avoid the more serious and complicated operation of tracheotomy. An intubation tube was therefore inserted, with complete relief to all dyspnea. Following this, however, difficulty was immediately experienced in extubation. The tube was no sooner removed than it had to be immediately replaced, the replacement always being accomplished with more and more difficulty. After several such experiences, it was feared that permanent trauma and possible stenosis of the larynx might occur, and it was decided to perform a tracheotomy. Thereafter, aside from some delay and difficulty in decanulation, there were no further symptoms.

In this instance, postoperative laryngeal edema did not prove as amenable to intubation as we had hoped. At least the tube facilitated tracheotomy, which was done with the intubation tube in place, as a guide. Whether the two bronchoscopies at intervals, however short, were more conductive to laryngeal edema than if both fragments of nut had been removed at one sitting, even with two introductions of bronchoscope, is an open question.

Failure to remove a known tracheal or bronchial foreign body has been the disappointing experience of most bronchoscopists. Under such circumstances, tracheotomy, if not actually imperative, may prove a last but successful resort. Either the foreign body may be coughed up thru the wound, or a second bronchoscopy thru the wound may be crowned with success. This is certainly not because one is working at any shorter range than thru the mouth, and yet, to the occasional operator, the avoidance of the introduction of the tube tru the larynx makes the operation at least apparently easier. Illustration of this situation is the case of a child of two years, from whose trachea we failed to remove a kernel of field corn on the first bronchoscopy in another hospital. The patient was immediately transferred to the Children's Hospital, where tracheotomy was done for impending laryngeal obstruction, postoperative in origin. In spite of this, great difficulty was encountered in maintaining a clear airway. The tracheal wound was enlarged and the tube finally left out altogether,

but the rapid formation of dry secretion and crusts constantly hindered us. It was finally decided that our only chance lay in removal of the corn kernel, which was accomplished quite easily by introducing the bronchoscope thru the tracheal wound. Following removal of the corn, the greatest difficulty was encountered for three days in keeping the tracheal airway free from crusts and inspissated masses of tenacious mucus. Repeated suction, swabbing and mechanical removal were required before expectorants finally restored these secretions to their normal fluid consistency. This tendency to crust formation in tracheotomy cases usually occurs only in the acute infections, such as described above, but in either type of case, it presents a complication almost as difficult to handle as the original obstruction. Hence, if it be true that in some of these patients, as claimed by pediatricians, intubation will avoid the open trachea and satisfactorily handle the laryngeal obstruction without untoward sequelae, then it would seem that this should be tried more often.

Tracheotomy has yet another distinct field of usefulness in those cases in which external pressure on the larynx is producing the obstruction. One example will illustrate this point.

A boy of one and a half was admitted with a severe cellulitis of the neck, producing a brawny induration, intense toxemia, and a beginning dyspnea due to pressure on the larynx and treachea. The visible edema of the epiglottis gave warning of the probable obstruction that might take place at any time. Free incision in the neck was immediately made, but the problem as to treatment of impending laryngeal obstruction remained. So much local laryngeal edema made intubation seem inadvisable, and the likelihood of increasing obstruction finally led us to tracheotomy. A special feature here was the degree of deviation of the trachea due to the pressure from the cellulitis, so that the line of incision lay almost parallel to the anterior border of the sternomastoid muscle. Convalescence was uneventful. It might be argued, that following incision of the neck, the laryngeal swelling would have soon subsided, but there was no sudden evacuation of pus, and we feel that the additional procedure of tracheotomy had much to do with the satisfactory outcome of the case.

Finally, we wish to call attention to the possible difficulties to be encountered in reopening a previous tracheotomy wound. Scar tissue may produce such deviations in anatomic sym-

metry as to cause considerable trouble at a time when it is not anticipated.

A convalescent case of laryngeal stricture, from which the tracheotomy tube had been removed, returned with evidences of an acute pulmonary suppuration, possibly abscess. It was finally decided to try bronchoscopic aspiration, but on laryngoscopy, the laryngeal lumen was not quite sufficient to admit the bronchoscope easily. Mild laryngeal dilatation, similar to what had been many times carried out before, was then performed. In a very short time, respirations became labored and impending laryngeal obstruction threatened. Immediate reopening of the old tracheal wound was indicated. Owing to the dense scar tissue, this operation was fraught with the greatest difficulty. Thinking that the bronchoscope might aid in the location of the trachea, one more hasty attempt at introduction was made but without success. By this time the child was in extremely critical condition, and it may well be realized with what relief an escape of air was finally secured from the trachea. The tube was replaced, all obstruction was relieved, and in a short time all evidences of pulmonary suppuration had cleared up, possibly due to drainage thru the tracheotomy tube. One may well bear in mind that a secondary tracheotomy will prove more difficult than the first one.

These then have been a few of our outstanding experiences with laryngeal obstruction. They have taught:

(1) That in severe laryngeal obstruction, immediate tracheotomy is the wisest procedure.

(2) That intubation, particularly in the noninfectious cases, may be a better means of relieving obstruction than tracheotomy, and

(3) That in the presence of laryngeal infection, relief of the obstruction alone may not suffice to prevent extension downward to the lungs.

DISCUSSION.

Dr. H. B. Orton, Newark, N. J.: It is unfortunate that Dr. Richards has had a bad run of cases. We are bound to have failures, and Dr. Richards has had some, and it takes a lot of courage to report them.

As far as using intubation instead of tracheotomy in draining the lung after a foreign body case, especially those of vegetable origin, is concerned, I do not think you will get very far with an intubation tube. Certainly it will not drain the lung if a tracheotomy tube will not.

In dealing with a staphylococcus laryngotracheal bronchitis, the mucus is so tenacious, that in a number of cases we have had to go down with a bronchoscope, the patient being relieved for a few hours, possibly twelve. Then it is necessary to go down again, in spite of which the patient ultimately succumbs, not to the tracheotomy or infection from it, but to the bronchitis that has blocked the terminal bronchi completely.

Of course the ultimate object of all this tracheotomy and intubation is to put air down into the lungs. It is also a fact, that after a tracheotomy or intubation, somebody must be there all the time, and these patients must have constant nursing for a considerable period of time. You cannot put these cases in a ward and expect the ward nurse, with all the rest of her duties, to look after the case properly.

Dr. Gabriel Tucker, Philadelphia, Pa.: I wish to agree with Dr. Richards, so far as our experience is concerned, as to the effect of tracheotomy in rest of the larynx. It seems to us that the indications for tracheotomy are, first, relief of laryngeal dyspnea; and second, drainage of the lung. In Dr. Richards' case, this was demonstrated, particularly in the last cases, where he drained a large lung abscess and nothing further was required. In cases of organic foreign body, where there is stenosis, usually there is marked inflammatory reaction, and the introduction of the bronchoscope adds something to the trauma. In these cases, the dyspnea usually becomes worse, and the question is whether we shall do an intubation or a tracheotomy. The thing that makes the dyspnea worse is drainage. When you remove a foreign body, a lot of secretion is liberated, and the dyspnea is progressively greater until we get the secretion thru the narrow glottis. When you put in an intubation tube, you put in a smaller lumen for this to go thru. Another thing, you put a foreign body into the larynx, which keeps up the irritation that we have in these cases, and it is better to do a tracheotomy, put the larynx at rest, and aspirate the secretion. We find, when we go into the trachea, the first thing that appears is a large mass of thick nasal secretion, and you will be surprised when you put in a tube to find that the patient breathes thru his mouth. It is not a question of laryngeal stenosis, but a question of deficient drainage of the field. At the same time, we have encountered a number of cases, with some failures, as Dr. Richards has had, and the reason is, that in some cases this secretion dries and forms crusts. In one case reported at the Clinic, seventeen removals of these crusts were required in order to keep the patient breathing. Recently we have had a number of these cases in which we have tried instilling normal saline solution with a medicine dropper—eight or ten drops—and we have been able to aspirate them easily. Use eight or ten drops of normal saline, and then aspirate and reaspirate, and

you will find you get the secretions from the smaller bronchi. In this way we have been able to pull several of these cases thru. This is particularly true in small children, under six months of age.

Subcutaneous emphysema following tracheotomy we have had occur in many cases, but without serious import. Dr. Richards, I believe, uses the same technic as we do, and putting the head back so the trachea comes forward is, we feel, best. We have, however, seen serious results from emphysema, but not subcutaneous. Several cases extended to the mediastinum; one or two succumbed to the emphysema, one particularly, that had a bilateral pneumothorax. That case, prior to our examination, had had a subcutaneous emphysema. There are several explanations why this occurred. Now we take the bronchoscopic field when a foreign body case has already had a subcutaneous emphysema. It is possible some of these cases of Dr. Richards may have had this condition.

Dr. Sam E. Roberts, Kansas City, Mo.: I want most heartily to agree with the early tracheotomy following a bronchoscopy. I think the dictum laid down by Dr. Richards, Dr. Jackson and Dr. Tucker is absolutely correct, i. e., that we watch for signs of respiratory embarrassment and not cyanosis. I am sure that in at least one of Dr. Richards' cases, he saved a life by that procedure; on the other hand he had some failures after tracheotomies in the acute infections. I want to make a plea for more conservatism in cases where there is violent, acute infection present. Any surgery at this time had additional hazards. In 1924 and 1925, I saw two of these cases in consultation. A tracheotomy had been done, and both proved fatal. I have also seen a number in which the indications appeared the same for a tracheotomy; it was not done and the patients survived. I would strongly suggest repeated intratracheal injections of some of the organic salt solutions. The frequency of these injections should depend upon the condition of the patient. This stimulates a cough reflex and will assist the patient in clearing the air passages. In the distinctly membranous types, this is especially important.

Dr. Samuel Iglauer, Cincinnati, Ohio: The great mistake made about a tracheotomy, not only by the general practitioners but by the laryngologists, is that often we postpone operation too long, and when we finally decide to do the operation, the patient is in extremis from exhaustion if not from asphyxiation. There is no operation which can be done with greater assurance than a deliberate tracheotomy, where you have plenty of time to get local anesthesia. On the other hand, where you have an urgent case in a small child, and have to do a stab operation, it is exceedingly difficult. In that case, you have to play bogie golf in order to get into the trachea.

The question of papilloma of the larynx was mentioned by Dr. Richards I agree that tracheotomy for papilloma is very valuable, and I think we have all had the experience, that after the tracheotomy, the papilloma often tend to disappear gradually.

The question of intubation was brought up, and I believe that Dr. Richards prefers tracheotomy to intubation in infectious cases. I think, however, that it is really better to do an intubation when possible. A case that presents severe infection of the throat and larynx may die of wound septicemia from the site of operation. One thing about an intubation, is that it is never safe to leave an intubated patient alone, and

I believe that unless you have a trained intubator in the hospital, you should protect these children by suturing the tube into place.

As to the question of direct examination of the larynx, if possible you should make that examination before undertaking the tracheotomy. Recently, I had a child with symptoms of croup recurring at night. Upon examination of the larynx, I found a piece of bone impacted in the larynx that had been there a week. Obviously, without direct examination, a tracheotomy would have relieved the case, and an intubation would have pushed the foreign body farther down.

The question of bronchoscopy before tracheotomy is important. By introducing the bronchoscope before doing the tracheotomy, as advocated by Gill, you can cut down on the bronchoscope with great assurance.

One will encounter all sorts of cases, and I would like to mention the case of a woman weighing about 300 pounds, who was brought to the contagious hospital with a diagnosis of diphtheria. Tracheotomy was indicated, but she was so fat that we could not put her on the operating table, so the operation had to be done in bed, thru great thicknesses of fat. We finally succeeded in getting into the trachea and she recovered.

Another woman was brought into the hospital in the ninth month of pregnancy. She, too, had diphtheria. We did a tracheotomy, and she soon went into labor. As a laryngologist, I advised the obstetrician to use forceps to deliver the child, but he said the uterus was sufficiently powerful to deliver the child without them. He did not realize, that in order to bear down, the woman had to close her larynx. She went into labor, but the labor did not proceed. Finally the interne, contrary to instructions, went in with forceps and delivered the child.

The after treatment is important. In accordance with the practice of Dr. Tucker, saline solution has been in use in the Cincinnati Hospital for some time. We instill the salt solution to loosen the secretions, then put on the suction pump to take them out, and after that, sometimes introduce sterile olive oil to prevent crust formation.

The question of emphysema is interesting. Some years ago, I reported a case of pneumothorax following tracheotomy. I looked up the literature and found that mediastinal emphysema and pneumothorax may occur after tracheotomy, but mine was the first case reported clinically, because I had an X-ray taken to look for a foreign body and found the pneumothorax. Experiments on the cadaver have shown, that with an obstructed larynx, air may be sucked into the mediastinum thru a small opening in the tracheal fascia. Therefore in tracheotomy, the fascia should not be nicked, but should be fully incised when exposed.

Dr. Frank R. Spencer, Boulder, Colo.: Personally, I am in the habit of doing a tracheotomy for several classes of patients: First, for children with obstruction caused by diphtheria; second, for tumors, either benign or malignant; third, for foreign bodies; and fourth, occasionally for advanced laryngeal tuberculosis. I would like to urge the importance of early tracheotomy. I believe I have had far more fatalities from too late tracheotomy than I have from early operation.

I have always been interested in the instruments Doctor Jackson has devised. I do not believe we can emphasize too much the importance of using the bronchoscope to aspirate secretions from the tracheobron-

chial tree, as in many instances we thus avoid a tracheotomy. If you have the patient in a hospital, you may try some things to avert a tracheotomy, but certainly you should not wait until it is an emergency operation. These patients are so much more likely to die before we have really had a chance to know what should be done for them.

Dr. Lyman Richards, Boston, Mass. (closing): In mentioning the case which had a tracheotomy for possible lung abscess, I did not intend to convey the idea that that would be a satisfactory method of treating pulmonary infection, but in that case, the pulmonary infection seemed to clear up after the tracheotomy, and it occurred to me that it might be because of the better drainage.

On going over the bacteriology in these cases, I assumed they were streptococci. Cultures were made, and in most cases they were streptococci.

I cannot stress too strongly the ordinary value of early tracheotomy. Before treating these cases, I had been an advocate of early tracheotomy, and the reason the question of waiting has been raised, is because in many cases, an early tracheotomy did not seem to give us results. The pediatricians have sometimes felt that I did my tracheotomy too early. That led to the question of intubation as at least a preliminary to tracheotomy. The objection that the intubation tube might easily become plugged by secretion is not a valid one, according to the advocates of intubation.

I have tried a variety of supportive measures after tracheotomy, and by using an oil, we have been able to keep some of these cases free from dried tracheal secretion; but once this secretion starts to cause bronchial obstruction, it is almost impossile to save these patients even by the earliest tracheotomy.

FIVE YEARS EXPERIENCE WITH THE HALLE OPERATION FOR ATROPHIC RHINITIS.

A. J. Lorie, M.D., and Paul Lux, M.D.

KANSAS CITY, MISSOURI.

Atrophic rhinitis is one of the easiest conditions in rhinology to diagnose, and one of the hardest to cure. I know of no condition that has had as many treatments promulgated for its cure, with as many failures. It is not within the scope of this paper to discuss the dozens of methods of cure put forth by our colleagues, but to simply state our experiences with the operation for the relief of atrophic rhinitis, advocated by Max Halle, of Berlin, in 1917. This operation has had few advocates in this country, due, we feel, principally to the fact that it has been insufficiently understood, and when attempted, the original technic has been so modified as to defeat the object in view.

Atrophic rhinitis, or ozena, is a condition associated with atrophy of the nasal mucous membrane and of the pharynx. With this is a destruction and atrophy of the underlying bony structure. These two conditions bring about a formation of crusts with a very offensive odor. The odor is generally believed to be due to decomposition of the secretions by the action of saprophytic bacteria. The diagnosis of this condition is so simple, that it is not necessary to discuss it further than to say, that the main points of which the patient complains, are: Difficult nasal respiration, due to the formation of crusts; intense dryness of the nose, due to an atrophy of the mucous membrane and to a disappearance of the glands which normally lodge in the turbinates; loss of sense of smell; and, in the later stages of the disease, a more or less disgusting and sickening odor.

The etiology of the disease is still obscure. There have been numerous theories regarding its causation, ranging from syphilis to endocrine disturbances, none of which have been proven. The pathology has been definitely worked out by Herman Sternberg, of Berlin, in 1923, and both the pathology

and etiology have been sufficiently discussed by the authors in a previous paper. (Amer. L. R. and O. S. Annals of Otology, Rhinology, Laryngology, Dec., 1924.)

We wish to report the end results of our first thirty cases. All of these cases were operated under local anesthesia, both sides being operated at the same sitting. The second division of the fifth nerve was injected thru the cheek with one-half of one per cent novocain. The infraorbital nerve was injected also thru the same puncture wound. The sphenopalatine ganglion and the anterior ethmoidal ganglion were blocked with local applications of cocain mud. The lateral nasal wall, after being freed from crusts, was gently rubbed with cocain mud. The floor of the nose was infiltrated with novocain.

1. An incision is started just in front and below the anterior end of the middle turbinate, carried downward along the lateral nasal wall, in front of the inferior turbinate, to and across the floor of the nose, and slightly up the base of the septum. This incision penetrates the mucous membrane and periosteum thruout.

2. The mucous membrane along the floor of the nose is completely elevated. This elevation of the mucous membrane continues up the lateral nasal wall, thruout its entire length, about half-way up to the attachment of the inferior turbinate.

3. A flat chisel is inserted in this pocket, formed by the elevated mucous membrane, and a straight line is cut thru the bony wall into the antrum in an anterior-posterior direction to the posterior end of the antrum. A slightly curved chisel is next inserted at the anterior end of the bone cut and perpendicular to it. With this chisel, an "L" shaped flap of bone is completed. This perpendicular incision in the bone must come far enough forward to miss the lacrimal duct, and is carried up just in front of the anterior end of the inferior turbinate and about a third of the way to the middle turbinate.

4. A flat chisel is next slipped into the antrum thru this opening, and rests on the antrum side of the lateral nasal wall near the roof. Strong pressure is exerted and the lateral nasal wall is fractured over against the septum. If the antrum is found diseased, it can be cleaned at this stage.

5. Care must be taken that the upper angle of this osteoplastic flap is well fractured, as failure to have this angle free causes most of the failures in this operation. Without exerting pressure upon it, this osteoplastic flap should now be in close contact with the septum.

6. Where the remains of the inferior turbinate come in contact with the septum, these two areas are thoroly scarified. A middle sized Killian speculum is inserted into the antrum, with one blade pressing this bone flap tight against the septum. The antrum is now tightly packed thruout with iodoform gauze saturated with vaselin. This packing serves the purpose of keeping these two scarified areas in close contact. The same procedure is followed on the other side.

Postoperative Care. The success or failure of this operation depends upon two points: First, on having the upper angle of the flap, formed by the lateral nasal wall, sufficiently fractured; and, secondly, on the proper packing of the antrum. The primary packing of the antrum is allowed to remain intact for five days, is removed, the nose and antrum cleaned of secretions and pus by suction or irrigation, and the antrum repacked, beginning at the same upper angle and coming downward. A smaller amount of packing is used at all subsequent packings; the antrum is repacked every five days until the gap in the front wall has granulated closed.

This operation accomplishes the purpose of greatly reducing the size of the nasal chamber, first, by shifting the lateral nasal wall closer to the septum; and, secondly, by rolling up the detached mucosa from the floor of the nose. In fact the nasal chamber may become so small, and lead to such a marked stenosis, as to interfere with breathing. This condition is even to be desired, and the nose should be left in this condition of stenosis for several months, for there is always a certain amount of retraction of the lateral nasal wall with subsequent healing. If, after five or six months, the stenosis is marked, some of the adhesions can be cut, allowing more retraction to take place.

In the ideal result the interior of the nose shows a remarkable change. Instead of the absence of the inferior turbinate, it appears as if a new inferior turbinate has taken its place. Instead of the dead gelatinous mucous membrane, it changes to a red, velvety, moist and almost normal appearance. There appears to be a rebirth of the glands in the lateral nasal wall, due, in all probability, to an increased blood supply. In the vast majority of cases, the odor disappears, crusting is of a minimum amount, if at all, and, if present, is situated around the middle turbinate. We have had a few cases where the sense of smell has even returned. The pharynx and larynx

take their share in this change and resume more nearly their normal appearance, due to the fact that the blast of air is being lessened and the process of preparing it for reception by the lung is being resumed by the nose.

SUMMARY. From the study of the following tables we find, as do most observers, that a large percentage of these cases occur in women. This disease is a chronic process. An attempt is made to approximate the length of time our patients have had this condition. All patients with positive Wassermanns have been eliminated from this report. The fact that we had so many patients from foreign countries, and of foreign parentage, in our list may be due to the fact that twenty-eight of these patients came from a clinic in the foreign settlement of our town, inhabited mostly by Russians and Italians. We have not included in this list any patients operated upon in the last two years.

CRUSTING. When we speak of a cast of the nose, we mean crusting of the entire nasal mucosa as differentiated from the less severe type, where the crusting is over the inferior and middle turbinates and along the floor. The milder type has a crusting only along the inferior turbinate and along the floor of the nose.

ODOR. Practically all of our cases who have had a cast of the nose, have had an extremely offensive odor. The odor in the other two types of cases has been correspondingly less. We feel that we may safely draw a simile from this, and say that the greater the amount of crusting present, the greater the odor.

PHARYNGITIS AND LARYNGITIS SICCA. The more marked the atrophy in most of these cases, the greater the involvement of the pharynx and larynx. In three of our severe cases, the larynx itself was involved.

COMPLICATIONS. The first case gave us the most severe complication, which fortunately had a happy ending. This was an infection of the lacrimal sac, undoubtedly due to trauma of the duct. It was necessary to drain this sac from the outside. Cases of hemorrhage, we feel, were due to loosening of the packing. They were not serious enough to give us concern.

RESULTS. The greatest objection to the operation by its critics has been that the lateral nasal wall retracts and the adhesions fail to hold. This condition happened in six of our

cases, but it is a relatively simple matter to go thru your old incisions and make a second attempt to bring about the desired results. This retraction, we have always felt, is due to a failure of the ethmoidal angle to be sufficiently fractured. If the flap, at the time of operation, hangs free against the septum, the adhesions will usually hold. If the results are bad, as far as relief of the symptoms is concerned, the patient is never any worse off than before the operation, as is the usual case in most intranasal surgery.

TABLE I—SEX OF CASES.

Number of cases	30
Females	26
Males	4
Percentage of Females	86.6
Percentage of Males	13.3
Average age	28.2
Average age of Females	27
Average age of Males	36.5

The predominance of atrophic rhinitis among females has been universally observed. The above percentages correspond favorably with other observers. No satisfactory explanation of this phenomenon has ever been made, but it has been used as one of the arguments for believing that the disease is due to an endocrine disturbance.

TABLE II—DURATION OF DISEASE.

Longest duration	32 years
Shortest duration	9 years
Average duration	17 years

Wassermann, negative 100%.

This disease has been noted from early childhood and, of course, on account of its chronicity, it is present at any advanced age. It is impossible to do more than approximate the duration of the disease in any one patient, and the above figures are simply an approximation from the patients' views upon the length of time they have been troubled with this condition. The Wassermann is negative in all of our cases because we have rejected from our lists any patient who had a positive Wassermann.

TABLE III—NATIONALITY.

Russian	9
Italian	6
Swedish	2
German	5
Southern Europe	4
Americans	4

The disease is more prevalent among people of foreign birth, especially in Russia and the south central European countries. Practically all of our patients have been clinic patients, as it is rather rare to encounter the disease in private practice. Only four of our list of patients are native born Americans, the large majority coming from the western part of Europe.

TABLE IV—AMOUNT OF CRUSTING.

Cast of Nose	17
Mid. and Inf. Turbinates	8
Inferior	5

Seventeen of the thirty cases are of the advanced type, with a crusting involving the entire mucous membrane of the nose, so that it could be removed in a cast, revealing flat lateral wall of the most advanced type of atrophy. Practically this entire group of cases had the characteristic ozena odor, while the next eight are of moderately severe type, with marked crusting under the middle and inferior turbinates. The disease had not progressed quite as far as the first group. The remaining five had the principal involvement and crusting along the floor and over the inferior turbinate. This last group of cases can be designated as atrophic rhinitis and not ozena.

TABLE V—TYPE OF ODOR.

Extremely offensive	18
Offensive	8
Mild	4

One notes the relationship here of the character of the odor to the amount of crusting. The more extensive the crusting, the more foul the odor.

TABLE VI—INVOLVEMENT OF PHARYNX.

Marked	13
Mild	8
Very slight	9
Involving the larynx	3

The involvement of the pharynx and larynx depends principally upon the duration of the disease and the amount of atrophy in the nasal chambers. We have had three of these cases where the atrophy has extended down and into the larynx, causing the same condition of the larynx as that found in the pharynx. All of groups 1 and 2 had extensive involvement of the pharynx, the pharynx presenting the same dry, glazed mucous membrane with a dark tenacious crust over its surface.

TABLE VII—AMOUNT OF ATROPHY.

Inferior turbinate	18
Inferior and middle	22
Entire mucous membrane	18

All of the cases had an atrophy of the inferior turbinate. Over two-thirds had an atrophy also of the middle, while over fifty per cent had an atrophy of the entire mucous membrane. In the severe type the inferior turbinate had, to all intents and purposes, practically disappeared.

TABLE VIII—DATE OF OPERATIONS.

1922	8
1923	15
1924	5
1925	2

Oldest case in its 5th year.
Newest case in its 2nd year.

We have presented none of the cases operated upon in the last two years, and we have presented only the cases where it was possible to reexamine at frequent intervals. Many of our cases disappeared from our clinics, and we were unable to trace them. Eight of these cases are in the fifth year, fifteen in the fourth year, five in the third year, and two in the second year, which gives us a reasonable basis for passing conclusions upon the lasting efficacy of this work.

TABLE IX—COMPLICATIONS.

Case 1. Infection of lacrimal sac, rt.
Case 10. Severe secondary hemorrhage, ten days after operation.
Case 15. Secondary hemorrhage 5 days after operation, due to packing becoming dislodged.

We had three complications in this group of thirty patients. The first complications occurred in our first case, undoubtedly due to faulty technic, of injury to the lacrimal duct on one side, with a corresponding infection of the lacrimal sac. This patient made an uneventful recovery after drainage of the sac. Cases 10 and 15 had secondary hemorrhages, one in ten days and one in five days. These were both controlled by packing.

TABLE X—FAILURES.

Cases 4, 9, 28, secondary operation, both sides, for failure of adhesions to hold.
Cases 6, 12, 20, secondary operation on one side for same cause.

Three of the cases, nos. 4, 9, and 28, met with failure on both sides of the nose, due to the adhesions refusing to hold. This could not be due to faulty technic entirely, because one of the cases was our 28th case. Three other cases, nos. 6, 12, and 20, required a second operation of one side only. All six of these cases were reoperated on thru the old lines of incision, the lateral wall refractured and the old adhesions reformed, the convalescence being satisfactory, tho protracted. The failure of the adhesions to properly stick is usually due to one of two conditions: The failure to have the upper ethmoid angle sufficiently fractured, or due to improper packing of the antrum.

TABLE XI—PRESENT CONDITION.

Odor: 1922, 8 cases, absence of odor in seven cases; one very mild.
1923, 15 cases, absence of odor in 12 cases; 3 mild.
1924, 5 cases, absence of odor, 5.
86% freedom from odor.

The most brilliant result obtained is the absence of odor after five years. Of the eight cases operated in 1922, one had a very faint odor, the other seven being free from it. In the fifteen cases operated in 1923, there was an absence of odor in twelve cases, the odor in the other cases being very faint. The cases operated in 1924 and 1925 were entirely free of odor. There was an entire absence of odor in 86% of the cases, which we consider rather a good result in nasal surgery.

TABLE XII—RESULTS, CRUSTING.

		Cases
1.	Cast of nose	17
	Freedom from crusts	8
	Very slight crusting in region of middle turbinate	9
2.	Middle and inferior turbinate	8
	Slight crusting in middle turbinate	3
	Freedom from crusting	5
3.	Inferior turbinate	5
	Freedom from crusting	4

Freedom from crusting, while not as striking as the freedom from odor, shows a tremendous improvement. In the severe type, where there was a cast of the interior of the nose, we have an entire freedom from crusts in eight cases. The remaining nine showed a slight crusting in the region of the middle turbinate. This is not severe enough to be troublesome. The results in group 2 were better, as only three of these eight cases showed a slight crusting in the region of the middle turbinate; while the results of group 3 showed a still further improvement, tho only one of these cases showed any crusting.

TABLE XIII—ACCESSORY SINUS DISEASE.

Involvement of both antra	6
Involvement of one antrum	3
Involvement of the ethmoids	2
Percentage of accessory sinus involvement, 30	

Approximately 30% of these cases showed some pathologic involvement of the accessory sinuses, principally the antrum. This finding corresponds with the findings of Halle and others, and tends to disprove the old theories that atrophic rhinitis is the end result of a chronic suppurative sinusitis.

Conclusions.

We feel that the greatest benefits, as a result of this procedure, are:

1. In the freedom from odor.
2. The crusting is materially benefited in all the cases, and this result, while not perfect, is sufficiently good to give remarkable relief.
3. Intense dryness disappears and the discharge is lessened, but does not entirely disappear.
4. The operation is easily and quickly performed, is relatively free from danger, and complications are rare.

Our conclusions, at the end of five years' work with the Halle operation for atrophic rhinitis, are that the results have been sufficiently good, so that we feel justified in continuing this method of procedure as offering, in our hands, the best and simplest method for relief of this condition.

DISCUSSION.

Dr. Albert C. Furstenberg, Ann Arbor, Mich.: I believe Dr. Lorie properly introduced the subject, when in speaking of atrophic rhinitis, he said he knew of no condition that has had as many treatments promulgated for its cure, with as many failures. I believe our experience at the University of Michigan clinic has been similar to that of many others, namely, that the new treatments that have been promulgated from time to time have usually produced discouraging results. We have had a fair show in a rather large clinic to try most of the popular remedies of the day, but it was not until three years ago, when Dr. Lorie introduced the Halle operation to us, that we were willing to offer a treatment which gave some promise of being of service to patients suffering from atrophic rhinitis. We have performed the operation thirty-two times, and can agree with Dr. Lorie in what he has said in respect to the results obtained. The most striking effect is the disappearance of the odor. The patient's friends and relatives bear witness to this fact, and in several instances have observed a marked change in the mental attitude of the patient. The mental hebetude and depression, virtually amounting to melancholia in some cases, has disappeared. When assured by their friends that odor is no longer present, they gradually lose the self consciousness that formerly made them more or less social outcasts. While the crusts do not entirely disappear, there is a marked diminution in the amount of tenacious adherent secretion. This is probably due to the small caliber of the nares, with consequent decrease in volume of inspired air, altho it may be argued that the increased blood supply plays some part in reducing the nasal secretions.

Dr. Lorie has told us of the remarkable changes in the appearance of the interior of the nose when the ideal result is obtained. It is a noteworthy fact, that on probe palpation of the lateral wall of the nose following operation, one gets a soft, cushion like sensation in the nose

and mucous membrane, rather than the old resistant feeling which one experiences when the probe encounters the firm surface of an atrophic turbinate. When one studies a microscopic section, there is decrease of tissue of the submucosa, complete destruction of the glandular fibers and underlying bony changes, and it is hard to understand how any operation could possibly bring about a histologic reconstruction of the tissues involved in atrophic rhinitis. Such a pathologic change probably does not occur, altho we have studied the nasal mucous membrane both before and after operation and found pathologic changes almost identical with the above description. There is one microscopic change, namely, an increase in the number of mucous membrane capillaries following the operation, and in those cases it might be reasonable to assume that the increase in blood is in part responsible for the remarkable change that appears in the interior of the nose. The histologic picture is apparently not changed, but it is perfectly obvious that the nasal cavities undergo a marked improvement following the operation.

I do not believe the operation is without danger. It is occasionally accompanied by shock, and in several instances we have had rather profuse hemorrhage.

We have followed postoperative dressing, and not infrequently the patient develops considerable swelling and discoloration of the soft tissues about the nose and orbits. Four of our cases, in a series of 32, developed dacryocystitis, but the function of the lacrimal sac returned spontaneously without surgical interference.

We had one serious complication. This occurred in a negro, who developed, on the third day following the operation, a unilateral proptosis, which was obviously due to thrombosis of the ophthalmic veins. The condition resolved in two or three weeks, but left the patient with marked impairment of vision in one eye.

Notwithstanding such cases, our experience with the Halle operation leads us to agree with Dr. Lorie, that it is the only treatment with which we are familiar that brings about sufficient improvement in atrophic rhinitis to justify a continuation of this technic in our clinic.

Dr. Harry L. Pollock, Chicago, Ill.: I want to congratulate Dr. Lorie on his splendid results, which differ materially from the reports heretofore published. In the beginning, at the time Dr. Halle presented his paper, we tried it in a number of cases, but probably due to faulty technic the results were very poor. But just recently, while in Europe, I had occasion to see in Vienna some of the cases that Dr. Halle had operated. He came to Vienna and operated a number of cases, and those I saw were just as bad off as before the operation.

We have done a great deal of work on atrophic rhinitis, and I presented a paper last year on some of our results. I still believe that every case shows ethmoid involvement, and probably antrum and sphenoid, about in the proportion given by Dr. Lorie.

The doctor says it is an easy operation. Anyone who tries it will find it a formidable one, subject to a great many complications, as described by Dr. Furstenberg and other authors.

We reported a number of cases last year, in which we had better results than described by the author, because all of them had been absolutely

free from crusts and odor for about three years. The only symptoms the patients show is an occasional mucoid discharge.

We have a technic which is very simple, and that is, lessening the space in these atrophic cases. We think that the clinical symptoms are due to the large spaces which we have in the inferior portion of the nose. If we can narrow them by any safe method, we will accomplish results. We do that by the very simple operation of elevating the mucoperichordium of the septum and placing a small piece of perforated ivory under it. Within 24 hours, the patients leave the hospital with little or no reaction, and in three or four days there is marked improvement. Previously, we do a radical operation to take care of the sinus infection. The sinuses must receive attention first, because after the ethmoids are cleaned out many patients get better. About six weeks after this sinus operation, we implant these pieces of ivory, one on each side, and in a short time the patients are relieved of all their symptoms. Patients in whom the ozena was so strong that one could hardly stand to be in the same room with them, report that the odor has entirely disappeared. They have no odor or dryness of the throat. We showed some pictures last year in which there was an apparent hypertrophy of the inferior turbinate.

In a few of my cases, the ivory has sloughed out. Possibly, in doing the operation, I tore the mucosa, and if the ivory is exposed it sloughs out, or we take it out. But in every case in which it remained intact, the patients have been free from symptoms. The longest time which has elapsed is three and a half years. It is a very simple operation, and in my hands it has given us better results than in the cases reported by Halle.

Dr. John C. Boone, South Bend, Ind.: I have had no experience with the Halle operation, nor have I seen any cases that have been operated, consequently I cannot speak as to the end results of the operation. I am forced to accept the report of the essayist and his chief discussant. It would seem that the operation has for its effect about the same thing as one by Dr. Pollock with which we have had some experience as a matter of observation.

The etiology of atrophic rhinitis, of course, will have to be determined before we can get any definite remedies. That is true in all diseases. We do not know whether atrophic rhinitis has a hereditary feature or not. Possibly it has, altho from the fact that the Wassermann was 100 per cent. negative in the cases reported by Dr. Lorie, it may have no bearing on the case. That, however, does not mean anything. The Wassermann is very eccentric in its results.

It appears to me that the operation has only two purposes. One is the narrowing of the nasal passages, thereby producing some hyperemia in the mucosa of the nose. The other is drainage of the antra. That it could produce renewal of circulation about the mucosa and restore it to its normal histology, I cannot conceive.

If the Halle operation and the Pollock produces practically the same results, possibly my conclusions are correct. I would not be too positive about these cases, but in my opinion the operation drains the antrum only as the chief benefit. I have not seen a case of atrophic rhinitis that was not complicated by some of the sinuses being involved, gen-

erally the antra. It is possible that drainage produces the results reported.

Dr. Francis L. Lederer, Chicago, Ill.: I think we are all agreed that Dr. Lorie is to be congratulated upon the excellent end results he has obtained in his series, and knowing well the demands made upon the operator, not only as to operative technic, but also in the after care which is so important, his results are certainly worth considering.

However, it seems that this procedure as outlined by Halle would be perfectly feasible in the hands of such a dextrous operator as the author, but no other than a person of such ability has any right to carry out such a formidable procedure, because it is more than likely that even more complications, such as the essayist mentioned, would occur. The difficulties he mentions, such as splintering of the nasolateral wall, are not uncommon features of this operation even in the best of hands. The packing over of this wall to the septum, which has been denuded is attended by considerable difficulties. The wall must be held tightly over for a considerable length of time, and to get a firm packing is quite a painful operation to the patient. Even after such careful operative care, the chances are that the adherence of the septum to the nasolateral wall is not a permanent condition, for the observance of these cases over a period of time shows a marked tendency for retraction in the majority of cases, and the space again becomes enlarged as before. Even in some of the socalled good results, crusting was seen to go on in the upper straits just the same.

Lautenschläger suggested forcing the lateral wall over by means of a sublabial route (similar to the Caldwell-Luc) and keeping the wall over by packing the antrum. Seiffert (Berlin) modified this by suturing both nasolateral walls to the denuded or scarified septum by means of an aneurism needle or wire. This operation, too, is by no means without its objections, namely being too formidable.

The implanation of ivory, as described, into the septum to narrow the space is by no means new, for many other types of material, both autogenous and heterogenous, have been employed for the same purpose. However, I have had results and seen results which make me feel the advisability of using this simple mode of treatment in helping out these seemingly hopeless cases. By implanting these pieces of ivory, suitably measured and prepared, you will find that the crusting has a tendency to subside almost immediately, and the reaction is so slight, that it almost becomes a minor procedure, especially in comparison with the Halle and Lautenschläger operations. Also, I have found of late, that it becomes unnecessary to do a previous ethmoid operation in these cases.

From a personal experience with the implantation of ivory on both sides of the septum at the same time, I would strongly advise against it, because of the interference with the circulation of the septum, the cartilage being almost avascular, and therefore there is a danger of decubitus, sloughing and finally perforation. We know full well that ivory is a foreign body, yet it seems to be well borne by the tissue, as illustrated in one case where the ivory was exposed and I thought I would have to remove it; but in a short time mucous membrane grew

over it to cover it completely. The ivory is still in, and the patient is free from previous crusting and odor.

Dr. C. G. Wencke, Battle Creek, Mich.: I do not want to take up your time in discussion, but I want to ask Dr. Lorie a question, and that is, where he has definite involvement of the posterior accessory sinuses, does he handle those before or subsequent to the Halle operation?

Dr. W. Likely Simpson, Memphis, Tenn.: Some little time ago, I had an opportunity to see Halle's cases. I did not get the impression that his cases were not all greatly benefited. I saw old cases and new cases. Certainly I had to take his word for it, but I saw his history of the cases before operation, and they all seemed to be greatly benefited.

Personally, I speak from an experience with five cases. In my cases, the sinuses were first attended to, and then the Halle-Lautenschläger operation was carried out.

The inferior turbinate becomes very large, and in many of these cases looks like an ordinary hypertrophy, even tho it is quite small previous to the operation.

I think the operation should be done thoroly, and especially should the packing be done well. The walls should be thoroly packed over to the septum. Personally, I like to leave the packing in for ten to fifteen days before it is removed. Of course, if there is unusual swelling, it might be necessary to remove it earlier than that, but after four or five days, if the swelling is going down and the conditions are good, I leave the packing in as long as two weeks.

The operation has been called formidable, but it does not seem to me that it is nearly as formidable as many operations within the nose. It is done in a few minutes, and I have had no complications whatever. Of course I have not had the experience some of the men have had, but I see no reason why there should be any great number of complications.

Dr. A. J. Lorie, Kansas City, Mo. (closing): In a previous paper, I spoke of the mental attitude of these patients and the remarkable change that takes place. I think this result well worth the operation.

I spoke of this operation as relatively free from danger, and I think it is. In our experience in close to 50 cases, we had no serious complications, and while we do get a tremendous amount of postoperative edema in some cases, it subsides quickly.

I am discussing simply the end results of our experience for five years with the Halle operation. I am sorry Dr. Pollock does not get the same results we have, but in a previous conversation with Dr. Pollock, he stated he was not following the original Halle technic; he left the packing in only five days, and of course could not get results. We have to repack the cases for six or seven weeks.

The implantation of ivory and foreign substances under the mucous membrane is not new by any means, but has been tried and discontinued by most men, because the results were unsatisfactory. Dr. Pollock probably has some new method of using these implants that gives him the desired result.

While it is much simpler to implant these cases than to do the Halle operation, I still maintain that the latter is relatively simple procedure,

and it rarely happens that we are more than thirty minutes operating both sides. In a recent letter, Dr. Halle says he is sewing these cases at the present time, using heavy catgut. He sews thru the two lateral walls and the septum. Silver wire has been tried, but it was almost impossible to remove it.

Retraction sometimes takes place on both sides. We have had that happen in six cases, and these were reoperated. But even if retraction does take place, the results are usually good. I have never seen a case where the mucous membrane went back to the original condition.

Regarding accessory postethmoid disease, of course they have to be operated before; this could not be done afterwards.

As to leaving the packing in ten days or two weeks, I have never tried that. I should imagine you would have to use a gas mask to remove it.

MASTOID INFECTION IN THE INFANT.

GEORGE MORRISON COATES, A.B., M.D.

PHILADELPHIA, PENNSYLVANIA.

It seems well to preface this discussion by a definition of what is meant by the title, Mastoid Infection in the Infant. As some age limit for infancy must be arbitrarily set, I will consider that we mean that period between birth and the time when the ear and its accessory cavity, the antrum, has assumed to a certain extent the characteristics of the adult. This would place the age of infancy as from birth to two years of age, altho many developing adult characteristics are found before this limiting period is reached, and a great change has taken place between the middle ear of a few weeks' old, with its almost absent air space, its short, wide, patulous and horizontal Eustachian tube, its reduplicated folds of attic mucosa, its obtuse angled drum setting, lack of bony external canal and mastoid process and cells, except the relatively large mastoid antrum, and the adult type structure developing rapidly as the months go on. The infantile ear that must be considered here as presenting a different question of diagnosis, is the one just mentioned. It must be understood, that many mastoids well below the period limit of two years show a marked development of the mastoid process with pneumatization.

What is meant by the term Mastoid Infection? In the adult type, undoubtedly, it means sufficient infection of the mucosa of some or all of the mastoid cells, with or without bone destruction and thrombophlebitis of the blood vessels, to give rise to local manifestations of inflammation over the mastoid area, systemic signs that can be attributed to such an infection, or infection and destruction of mucosa and bone without recognizable signs or symptoms until a long interval of time has passed, or the autopsy has disclosed the seat of disease. In the infant, where the mastoid has not developed, we experience greater difficulty in defining this condition, since we have, by the terms of our previous definition, no mastoid to

deal with except the large antrum, or at the most a few surrounding pneumatized spaces. The mastoid is still diploic in type. Mastoiditis, therefore, to be distinguished as a separate entity from otitis media, must mean infection of the antral mucosa, of the bony walls of the antrum and of such few surrounding cells as may be present, sufficient to cause local symptoms calling attention to the localized infection, or general symptoms which may or may not be linked up in diagnosis with this local infection. Can there be present in the infant's mastoid infection sufficient to cause general symptoms of a serious or fatal nature, without attention being attracted to the mastoid or without the possibility, in certain cases, of making a diagnosis by observation and study of the mastoid? If so, is there a large group of such cases observable? Opinions differ widely on this matter, and as it seems a matter of some moment, it will be discussed thoughtfully and impartially later on.

With these two definitions in mind, let us examine briefly the anatomy of the infant's middle ear and mastoid. The Eustachian tube is relatively shorter, broader and more patulous than in the adult; the external bony canal is lacking, the mastoid process is undeveloped, diploic in character, with but one large cell, the antrum, and later a few small, surrounding air spaces. The mastoid cortex is usually thin, and the mucosa of the attic and antrum redundant, lying in folds. The middle ear and antrum are normally sterile, but for the first few weeks of life, at least, the mucous membrane more or less completely fills these cavities, and hearing is undeveloped until the fourth day. The inner table is usually thicker and more resistant than the cortex.

As, according to Gomperz and Alexander,[1] the infantile tympanic membrane is more resistant than in the adult, and as the folds of mucous membrane in the middle ear and attic prevent the early accumulation of a large mass of infected secretion, pressure upon the drum, with resultant necrosis and perforation, is often much delayed. This delay may be prolonged by drainage of infected material thru the wide patulous Eustachian tube, so that while general symptoms of infection may be apparent, there is little to direct attention to the ear or, frequently, little to see if examination is made. Since some of the diagnostic points of mastoiditis are those of middle ear in-

fection or its absence, the two can not be considered entirely separately.

The ordinary frank type of mastoiditis seen at an advanced stage offers no greater difficulties of diagnosis in the infant than a similar type in the adult. There is frequently the same history of acute rhinitis (sinusitis) and tonsillitis, an attack of one of the acute exanthems, and occasionally, the history of trauma, which may be a factor. The subject is more often debilitated and undernourished than otherwise. In the youngest cases the presence of pain cannot be ascertained, and in the older ones it can only be inferred by restlessness or scratching at the ears. The fever is from moderate to high, with some irregularity of curve, and the white count may show some leucocytosis. Bacteriology, in the typical case, according to Alden,[2] shows the infecting agent to be usually a pneumococcus, staphyloccocus or nonhemolytic streptococcus. This is the picture of otitis media as well, for otitis media of some degree is the almost necessary precourser of mastoid infection. If the mastoid is infected, tenderness to pressure may be demonstrated by gentle bimanual pressure with the fingers, first on regions well removed from the mastoid, gradually working down to the antra. Ersner[3] strips his babies before examination, as a kicking, unclothed baby is happier, less fretful and more likely to react properly to the pressure. At the same time, the soft velvety feel of inflamed periosteum can be noted and contrasted with the well side if the case is unilateral. Rumpling of the skin over the mastoid with the finger gives a sensation of attachment or rigidity on the affected side, contrasted with free mobility on the normal. Inspection from the rear should show the auricle displaced downward and outward in an advanced case, or reveal flattening or loss of one or more of the normal postauricular creases. Frank edema is diagnostic, if skin infection of the surrounding areas, including the external canal, pediculosis capitis, etc., can be excluded, and this statement applies also to the next stage—fluctuation—which then means cortical perforation. Inspection of the external canal shows quite as constantly as in the adult the drooping of the posterosuperior canal wall, but this manifestation of mastoid periostitis is more superior than posterior, because of the absence of the external bony canal and the subantral cells, and the slightly higher and forward position of the antrum. The external canal, being com-

posed solely of soft tissues, is frequently seen as a narrow slit, due, according to Alexander,[4] not entirely to swelling of the soft tissues themselves, causing the posterosuperior wall to descend, but also to passive stretching of the anteroinferior wall. This ellipitical canal opening extends, in its long axis, in a direction from anterosuperior to posteroinferior. Often the slit is so narrow, as the canal nears the drum, that it will not permit the passage of a speculum, and examination of the latter is imposible. In any case, only a small speculum can be used, and a self lighted one is usually essential. The child should be rolled in a blanket and held as immovable as possible on the bed, and the canal cleansed of cerumen, pus and debris. The tympanic membrane, if seen, will now present any of the usual characteristics—inflamed, bulging in whole or in part, and red, pink, or grey, perforated with discharge, or approaching normalcy.

Fever, present during the preceding otitis media, may be high, or absent, depending largely upon the amount of drainage present, the virulence of the infective agent and whether or not perforation of the membrana tympani or mastoid cortex has occurred. Leucocytosis of moderate degree is present, with a polymorphoneuclear percentage of from 70 to 80. The X-ray gives little information, since the area involved is too small—it gives no information as a rule unless the child is anesthetized with a few drops of chloroform.

Perforation of the drum often occurs late in the course of the otitis media, while perforation of the antral external wall, or cortex, takes place easily and quickly, often with relatively few symptoms.

We must remember that pneumonias, at least of a certain type, have been attributed by creditable observers to infections in the nasal accessory sinuses, or in the middle ear and mastoid, and certainly autopsies done on victims of influenza in the Army epidemic of 1918 disclosed a surprising number of middle ears and mastoids that contained purulent secretion. Was this the cause of death in these "flu" cases? Most of them, during life, were symptomless as regarded the sinuses and ears.

As far back as 1868, Wreden[5] observed, that at autopsy of year old infants dying of bronchopneumonia, the middle ears and antra showed evidence of inflammation or infection, but attributed this condition to the pneumonia and did not regard

it as the cause of the latter. These observations were confirmed by Rasch[6] (1897), who found inflammatory conditions of the ears in forty-two out of forty-three children having died of bronchopneumonia, and Ponfick[7] reported the same results in ten out of eleven autopsies. This situation is attributed by Friedrich,[8] Gomperz,[9] Alexander and others to the fact, that the undeveloped ear of infancy is especially prone to infection in any disorder which reduces the individual's normal resistance and interferes with nutrition, and they include in this category malnutrition, pneumonia, various intestinal affections, rachitis, tuberculosis, syphilis and others. Friedrich suggests, in 1900, that this process may be reversed, and that the ear infection may be primary and the systemic manifestations secondary.

This suggests the consideration of what must be considered an atypical type of middle ear and mastoid infection in infants, the diagnosis, at least as far as the indications for operation go, depending largely on systemic or focal reactions in other parts of the body, and to a lesser extent on local manifestations. As I have just pointed out, this subject and this controversy is not new, but it has recently been brought again before the profession by the reports of Byfield[10], Marriott[11] and Jeans,[12] pediatricians, Dean, Alden,[13] Lyman,[13] McMahon[14] and others, otologists, who have advocated closer study of certain types of infantile gastrointestinal disturbance in relation to the ear as a *cause* of the infection. Hartmann,[15] in 1894, called attention to the significance of middle ear and mastoid infection in nutritional disturbances of infants, and Goeppert[16] believed that the otitis was secondary to the intestinal disorder, and found that ear complications were so prevalent in this class, that only 12% of those admitted to hospital had healthy ears. Friedrich believed that much of this ear disease was secondary to intestinal disease and loss of resistance, but says "secondary nutritional disturbances play an important role in *primary* ear disease in the case of infants. A regular digestion and a uniform increase in the body weight afford the best criterion of an infant's health, for its digestive organs are so sensitive, that the slightest local or general disturbance may suffice to upset its stomach. If there is a purulent focus anywhere in the body, autointoxication will be much more likely to result from the absorption of the products of metabolism in the infant than in the adult, and will

show itself chiefly in the organs which are functionally the most important; that is, it will be followed by indigestion and a falling-off in weight." Hartmann[17] and Ponfick, in the Berliner Kinderklinik, found that often intestinal disturbances of an obstinate nature yielded promptly to the discovery and evacuation of infection in the middle ear.

Recent interest in this question was stimulated by the often quoted report of Renaud,[18] who, in a series of seventy consecutive autopsies on infants, found extensive suppuration of the middle ear and petrous bone in all, and in a large number such infection was bilateral. The histories of these cases did not show ear involvement, but they had been, for the most part, weak, undernourished children from poor hygienic surroundings who showed, on admission to hospital, a more or less acute syndrome manifested by emaciation, signs of infectious intoxication, fever, vomiting and diarrhea. No significant lesions were found in the intestinal tract. The spleen, lungs, liver and kidneys all showed signs of inflammatory reactions, and there was considerable edema and congestion of the vessels in the brain, hemorrhage and thrombi being found in some cases. He thinks that the diagnosis of gastroenteritis was never confirmed at autopsy, and that suppuration of the ear is the essential, if not the exclusive, factor in infant mortality.

Leroux[19] thinks that a *latent otitis* is the rule in every new born infant, and that the ear may be infected from the first inhalation. Kistler,[20] working in the Virchow Pathologic Institute years ago, noted the fact, that in a large number of infants, the mastoids were found full of pus in nearly every case. The history of these and the cause of death is not reported.

In view of these and many similar reports, the question at once arises whether these accumulations in the ear cavities are antemortem or postmortem in origin, and opinions will be found differing as widely on this subject as on all other points at issue. Alexander states that "after death the mucous tissue undergoes exceedingly rapid decomposition from putrefaction, filling the tympanic cavities of infants with a gelatinous, more or less purulent, fluid." On the other hand, McMahon, studying histopathologically the tissue removed during antrum drainage operations done by Lyman for the relief of gastrointestinal disorders, finds ample antemortem evidence of path-

ology, and Alden, Lyman and many others attest to the certainty of infection by cultural methods. This is the only way to prove the point—the mere presence of a purulent *looking* material at autopsy (possibly long delayed) is not conclusive.

The general consensus of opinion seems to be that middle ear (and mastoid) infection, from whatever cause, whether latent, masked or manifest, is of very frequent occurrence in infants, particularly in those with lowered resistance from chronic or acute disease, general malnutrition and unhealthy surroundings. Gomperz attributes this to:

1. The short, wide Eustachian tube and the position of the nasopharyngeal opening.
2. Changes in circulation following birth, together with hyeremia and loosening of the tympanic mucosa.
3. Persistence of the embryonal tissue of the middle ear mucosa beyond the fetal period (this tissue being especially subject to infection).
4. Immaturity and undeveloped resistance.
5. Heredity and constitutional taints—tuberculosis, syphilis, rickets.
6. Infectious diseases—sneezing, crying, coughing and vomiting, forcing infection into the ear thru the wide, short tube.
7. Hematogenous infection.
8. Gastrointestinal and cerebral affections in which vomiting exists.

The tympanic cavity of infants contains abundant mucous tissue and fluid, according to Alexander, which makes an excellent culture medium for invading bacteria. This myxomatous tissue, embryonal in type, lies in many redundant folds, in great quantity, and is subject to rapid purulent transformation. The accumulation of this tissue, with connective tissue bridges, fills the epitympanum and much of the mesotympanum; the folds are thick and succulent, and when invaded by infection, there is not at once one large cavity containing pus, but many small ones communicating with difficulty, rarely causing perforation of the drum during the early stages for this reason, but offering ample chance for absorption by lymph and blood vessels, and to a less extent discharge into the stomach and intestines thru the wide Eustachian tube. Bryant[21] states that the lymphatics are nine times more permeable than in the adult, and therefore nine times more capable of

transmitting the products of infection. The mucosa of the mastoid antrum and aditus conforms in general to that of the middle ear, and closure of the aditus by swelling of infected mucosa may easily take place, and may well leave infection trapped in the antrum after the infection of the tympanic cavity has subsided. Absorption from such a latent focus may readily be accountable for focal symptoms with little evidence of local trouble.

Wide differences of opinion exist among both pediatrists and otologists as to the cause and effect relationship of gastrointestinal affections and otitis, just as they existed thirty or forty years ago. Possibly the solution is nearer at hand than many think.

It is in this class of cases, namely, middle ear and mastoid infection associated with systemic manifestations in other parts of the body, either in the respiratory or gastrointestinal tracts, that the present day problem of diagnosis is difficult. What is this association—cause or effect, *post hoc* or *propter hoc?* The recent notable work of Alden, Lyman, Dean, Byfield, Marriott and Jeans in the West has caused great renewal of interest in this question, and in some instances, alarm lest overenthusiasm lead to indiscriminate operating without due and careful study and diagnosis in the individual case. And opinion seems to vary sharply, depending on geographic location. When these eminent men tell us that they believe the socalled cholera infantum syndrome, and other intestinal manifestations, are caused largely by tympanic infection, great respect must be paid to their opinions, since they are reinforced by many detailed case reports, showing gain in weight, lowering of fever and cessation of diarrhea when the middle ear or mastoid antrum is drained. They have seen many such cases in a relatively short space of time, and in consequence the mortality from these affections has been reduced, by their method of treatment, to a marked degree, many of these cases formerly being considered hopeless.

On the other hand, Eastern pediatricians tell me that, since the rigid municipal supervision of the milk supply and general improvement in the hygienic and sanitary conditions of the home, very few such cases are seen by them either in private or hospital practice. The Chief of Pediatric Service in the largest hospital in Philadelphia tells me that he sees but one or two such serious cases in a year, whereas formerly he

saw many. A leading New York pediatrician and author says the same thing, and that his cases of malnutrition, athrepsia, dehydration, etc., yield to nonotologic management. The opinion of these men is also entitled to respect. Is it possible that we are dealing with and talking about similar conditions with different etiologies? Jeans (quoted by Dean) has suggested that this might be so, and that the climate of Iowa might be responsible for the apparently greater prevalence of such infections there.

Otologists, also, in other parts of the country have been rather inclined to look askance on the midwest practice, being influenced by their pediatricians and seeing few cases, altho few otologists of experience anywhere cannot recall gastrointestinal cases, or cases with fever and malnutrition of obscure origin, that cleared up promptly after a myringotomy on an almost normal appearing drumhead. Such instances must give us pause, but that a bilateral mastoid operation should be performed by the otologist on apparently normal ears, on the simple demand of the pediatrist, as I have heard recommended in open meeting is, I think, dangerous to the reputations of both specialties, to say nothing of the welfare of the patients.

Is it necessary or practical, then, for the pediatrician to make the diagnosis for the otologist? What are the otologic findings that can be considered diagnostic in these cases of atypical or masked infantile mastoiditis? If there is aural discharge, a bulging drum, mastoid edema, etc., the case comes under the heading discussed at the beginning of this paper. No one questions the necessity of a drainage operation under these circumstances. Aside from these, the trained otologist, examining carefully with brilliant light in a magnifying otoscope, will find certain guiding signs of infection if it is sufficent to cause the syndrome. Slight discoloration of the drum, a change to a grayish color with loss of luster, giving it a ground glass appearance; slight fullness, or redness, particularly of Schrapnel's membrane, slight posterosuperior canal wall sagging; these are all that may be found. Alden states that the infecting organism in these cases is almost invariably the streptococcus hemolyticus. Are these enough to enable us to make an operative diagnosis? Yes, when combined with continued fever, loss of weight, dehydration, diarrhea and vomiting, otherwise unexplainable; no, decidedly, when these,

or similar, general symptoms are not present. To say that every case of infantile diarrhea with slight fever of but short duration should have a double mastoid drainage, in spite of the absence of all otologic signs, is of course, *reductio ad absurdum*; and yet that is precisely the attitude that is being held and discussed by pediatricians and otologists in certain portions of the country. Those holding this view need the curb; possibly, also, those who are entirely opposed need the spur to seek more carefully for otologic reasons for the few cases that they report seeing. No one will dispute the dictum, that every infant, with or without some signs pointing definitely to the ears, who has fever, diarrhea lasting more than a day or so, any nutritional disturbances or respiratory symptoms should have a careful and thoro ear examination. If ear infection is found, the prompt institution of ample drainage is good practice, and if a myringotomy with free drainage fails to relieve the situation, postauricular drainage is indicated, for copious, prolonged, increasing, or at least nondiminishing, aural discharge is in itself indicative of mastoid involvement.

Altho the question of operation, whether myringotomy or antrotomy for drainage, is somewhat outside the scope of this paper, it has seemed to me, that in grave cases, operation adds little to the hazards of the case. I have for years taught my students, that in case of doubt, it is better to drain where systemic symptoms are grave, even when local ear manifestations are inconclusive; and we have all seen striking results from this procedure, which apparently demonstrates that the ear infection is, at least in many cases, primary. We should, however, subject our cases to the closest possible study and scrutiny, and be constantly on the watch for means of improving our methods of otologic diagnosis in these cases.

There are then a number of hypotheses open for consideration in these cases of systemic infection with apparent mastoid involvement.

1. Is the condition of the mastoid found at autopsy in the series quoted due to postmortem changes?

2. Is the middle ear and mastoid infection secondary to the systemic infection, and, if so, is it due to a consequent lowered resistance or to forcible injection of infected material thru the Eustachian tube, or to both?

3. Are there different types of cases of different etiology

with serious gastrointestinal symptoms in different parts of the country?

4. If serious or fatal gastroenteritis is caused by middle ear and mastoid disease, should not cases of infantile pneumonia and bronchitis be given equally careful study and examination, with the expectation of discovering a similar etiology?

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DISCUSSION.

DR. A. M. ALDEN, St. Louis, Mo.: It is always a privilege to hear Dr. Coates discuss any otologic subject, but I have especially enjoyed this paper, because for several years I have been deeply interested in this phase of otology.

In 1915 and 1916, at Hospital Santo Tomas in Panama, the middle ears of one hundred babies coming to the autopsy table were examined bacteriologically. The exact causes of death of these infants are not available at the present time, but in Panama gastrointestinal disturbances and bronchopneumonia account for most of the mortality in babies under two years of age. In this above series, forty-three of the babies had apparently normal and sterile middle ear cavities. Fifty-seven of the cases had demonstrable suppuration, and cultures showed staphylococcus to be present 26 times, pneumococcus 17, streptococcus 31, colon bacillus 2 and bacillus pyocyaneus 1. For this reason, I do not believe that the apparent pathology observed at autopsy in the middle ears of babies by Renaud, Leroux, Kistler, Rasch and others is due to postmortem change, but I am sure that it is there as a result of infection which existed during life.

That ear suppurations in infants are much more common than we had hitherto supposed, we now know. That there exists in many babies a definite causal relationship between ear infection and certain forms of gastrointestinal diseases, and that adequate treatment to the ears often produces an immediate amelioration of the gastrointestinal syndrome, has been demonstrated repeatedly.

To the trained observer, a middle ear suppuration, even in an infant, is never present without macroscopic changes in the fundus of the ear canal. Due, however, to the various anatomic factors which Dr. Coates has mentioned, the indications for paracentesis in an infant are somewhat different than in the older child or adult. In the baby, an ear drum that is lusterless or yellowish in appearance is often the only sign of a suppuration filled middle ear. Postauricular drainage is never done upon these cases without definite otologic indications, and even then, only when, in spite of all conservative treatment, the ear suppuration continues unabated and the general condition of the baby as shown by the fever, diarrhea and weight curve is becoming progressively worse.

Just what relation exists between ear infections and bronchopneumonia is at the present time problematic. The existence in the same child of a chronic otitis media or mastoiditis and a maxillary sinus suppuration on the same side, occurs too often to be explained upon the basis of pure coincidence. We know that a definite causal relationship often exists between paranasal sinus infection and disease elsewhere in the upper respiratory tract. Is the local spread of the infection in these cases the result alone of direct contiguity and gravity, or is there some other pathologic mechanism at work?

Another question that seems to come naturally at this time involves the possible relationship between these obscure middle ear infections in babies, many of which must heal spontaneously, and the socalled non-suppurative or catarrhal forms of middle ear disease, which become

manifest later on in life. Is the one a precursor of the other? I suggest this problem as a suitable one for consideration by the Research Committee of the American Otological Society.

The answer to these questions will probably come only as the result of careful collective investigations, and a better comprehension of the pathology of the infections involving all of the various portions of the upper respiratory tract will certainly lead us closer to an understanding of these very important disease relationships.

The note of caution voiced by Dr. Coates lest overenthusiasm based upon the results obtained by this procedure in properly selected cases lead to unnecessary surgery upon babies already in a debilitated condition, is indeed a propos. When our work on this subject was first reported, our conclusions were received with considerable skepticism on the part both of otologists and pediatrists. The criticism was made, that what we advocated was radical, that it was not good otology, and that it would probably lead to much indiscriminate operating being done upon infants. As to the first criticism, it need only be said that a life saving measure is never radical. As to the statement that our method of treating these mastoid infections in small babies is not good otology, I am sure that nothing that we have said in any of our papers contains any statement or suggestion that runs contrary to the fundamental precepts laid down by our otologic forefathers. We are simply giving the baby the benefit of the same standard of otologic diagnosis and therapy that our older patients have received for many years. In our previous contributions upon this subject, we have laid down fairly definite and clean cut indications for operative interference, and I am sure that if these indications are followed, unnecessary surgery will be avoided.

Dr. Frank R. Spencer, Boulder, Colo.: Doctor Coates, as usual, has left few points for discussion. Two points I should like to emphasize. First, the importance of the notch of Rivinus, the point at which the pus may pass from the attic up under the periosteum over the mastoid. Doctor Coates mentions the fact that pus collects there before we have much bulging of the membrana tympani. This pus may collect under the periosteum and behind the auricle. In that case, the antrum should be opened in order to have complete drainage and a cure.

Another important point is the superficial location of the seventh nerve when there is infection of an infant's mastoid. If an attempt is made to open the cells of the tip, there is very great danger of injury to the facial nerve. But in the infant, there are practically no cells in the tip, so the nerve can easily be avoided.

Dr. H. W. Lyman, St. Louis, Mo.: Misapprehensions regarding the procedure under discussion have clouded the issue materially.

Gastrointestinal disturbances in infants may be due to direct enteric infection, to food poisoning or to some parenteral infection. This parenteral infection may be in the middle ear or mastoid, and it is this type of case that we have under consideration.

The otologic picture, as both the essayist and Dr. Alden have said, is clear cut, and is the basis upon which we place our otologic service.

I have three slides I would like to show, pointing out some of these features.

Dr. George M. Coates, Philadelphia, Pa. (closing): I am very happy to say that I have no fight with Dr. Alden and Dr Lyman and their coworkers; I am entirely in accord with them. I first went into this largely because I had been misquoted and misapprehended. The impression has certainly gotten around in a large part of the country, that we advocate bilateral antrum and mastoid drainage in every case of malnutrition and diarrhea. It is to combat this idea that we must all work. Certain cases of constitutional disease and of gastrointestinal disease seem undoubtedly due to otitic causes. How can we determine which cases are due to that? There are cases that are not due to ear disease. When pediatricians make that diagnosis, they must rule out the other class of case, and when that is ruled out, there is no question of otologic diagnosis.

While these cases have appeared in great numbers in some parts of the country and not in others, and while the results in certain portions of the country following antrum drainage have been good, they have not been good in the hands of other surgeons. I talked to a Boston surgeon who said he had several cases in the past year in which he did drainage, and he did not get any results at all. I am entirely unable to explain why in St. Louis and other places they should get a large percentage of recoveries, and in this instance no recoveries at all.

Dr. Spencer spoke of the case with which pus may travel from the mastoid operation thru the notch of Rivinus. That is true, a difference in the sagging of the drum in the posterior superior wall in infants having been shown.

In doing myringotomies in these cases, with the incision of this sagging mass, we make the incision high, frequently to the middle ear, and at the same time the mastoid antrum receives a certain amount of drainage. We must not forget in making a diagnosis, that frequently the infection is not in the mastoid, but in some of the nasal accessory sinuses. When the infection comes originally from the accessory sinuses of the nose, if we pay attention to the original focus, the ear condition will be much relieved.

AVENUES OF INTRACRANIAL INFECTION FROM THE NOSE: PARTICULARLY FOLLOWING NASAL SURGERY PROCEDURE; ALSO RELATION OF EXTERNAL INFECTION TO INTRACRANIAL COMPLICATIONS.

(Lantern)

By

John A. Pratt, M.D., F.A.C.S.

MINNEAPOLIS, MINNESOTA.

There are six groups of intracranial pathways of infection.

1. Possible Embryonic Dehiscences: In the embryonic development, there are possible ways of leaving a dehiscence that might be a pathway of infection to the meninges. The cribriform of the ethmoid is cartilaginous at birth and does not ossify until the second year. The crista galli ossifies between the second and third years, while the lateral masses, which form the roof of the ethmoid capsule, are ossified at birth. Complete fusion of different parts of the ethmoid does not take place until the sixth year, and ossification not until the sixteenth year. The ethmoid bone, however, becomes of adult size by the seventh year.

Exenteration of the ethmoidal cells on patients under sixteen years of age, with removal of the middle turbinate, would be a very dangerous procedure, but with the middle turbinate left in place, a safe operation.

The basipharyngeal canal, which extends from the sella turcica thru the sphenoid bone to the pharynx, is really a part of the embroyonic inpush of the Rathke pouch, and is still open in about five percent of the cases at birth. It could remain open as an embryonic dehiscence from the sphenoidal sinus, thus accounting for meningitis from sphenoidal infection that leaves no trace of the pathway, especially if the cavernous sinus is not infected.

Middle Ear: The petrosquamous fissure, which passes thru the middle of the tegmentum tympani, might leave an

embryonic dehiscent pathway to the meninges. As shown by Kisselbach's report of three hundred and thirty-six cases examined, the ages ranging from birth to eighteen years, there is a gradual reduction of the closure of this fissure, of from 78.8 percent to 11.1 percent. Up to eighteen years of age, there is a possibility of eleven out of each hundred having a possible infection thru a dehiscent pathway from the middle ear. Thus the meningitis following infections of the nose, throat and ear during early life may be thru these embryonic dehiscences.

TABLE.

Obliteration of the Petrosquamosal Fissure in Childhood.
(Summarized from the data of Kisselbach).
336 cases.

		Condition of the Petrosquamosal Fissure			
Age	Number of Cases	Entirely Open (Percent)	Partially Closed (Percent)	Indicated by Pores and Clefts Course (Percent)	Entirely Closed (Percent)
Birth to 1 year..	52	17.3	78.8	0.0	3.8
1 to 2 years...	26	0.0	46.1	0.0	53.9
2 to 3 years...	46	4.3	35.6	0.0	52.6
3 to 4 years...	52	0.0	48.1	17.3	34.6
4 to 5 years...	36	8.0	31.5	13.2	47.4
5 to 10 years...	86	0.0	20.5	8.8	70.7
10 to 15 years...	20	10.0	23.3	6.7	60.0
15 to 18 years...	18	0.0	11.1	22.2	66.7

2. The pathway to the meninges may follow in any direction by continuity, due to destruction of tissue from malignant or suppurative conditions of the eye, ear, nose and throat. The radical operation on these parts eradicates the point of infection and tends to prevent the spread of the disease to the meninges.

3. The venous pathways are those taken by infections that seemingly start in the most simple way and lead to the most serious results.

The point of attack preceding the meningeal infection is the cavernous sinus.

The portion of the cavernous sinus first invaded depends on the venous pathway taken by the infection. Dr. Eagleton has named these as follows: (a) the anterior, (b) the anterior inferior, (c) the posterior inferior, (d) the posterior and (e) the mesial paths.

A. The current in the veins of the anterior part of the face normally drain into the facial vein and thence into the jugulars. Owing to the lack of valves in these veins, a slight blockage may deflect the current thru the communicating angular vein into the ophthalmic vein, then into the cavernous sinuses and the other sinuses of the head.

Infections of the face may, by venous circulation or retrograde thrombophlebitis, cause thrombosis of the cavernous sinus and eventually meningitis. Early destruction of the angular vein would block this pathway. This is called the anterior or ophthalmic pathway.

B. The pterygoid plexus receives veins from the deep tissues of the side of the face, the pharynx and the tonsillar region, emptying into the facial vein and then into the jugular. It also has communicating branches which empty into the ophthalmic vein and the cavernous sinus, and by these branches may carry infection into the cavernous sinus. This is called the anterior inferior pathway.

C. Any infection that would cause a thrombosis of the jugular vein could, by a retrograde thrombophlebitis, follow thru the bulb and the inferior petrosal sinus to the cavernous sinus. This is called the posterior inferior pathway.

The infrequent infections of the cavernous sinus from a thrombosis of the lateral sinus, is due to the angle of insertion of the petrosal sinuses. A descending infection from the lateral sinus obliterates these openings, while a retrograde thrombophlebitis of the jugular vein will infect it.

D. An ear infection that would cause a thrombosis of the inferior petrosal sinus directly is called the posterior pathway.

E. The osseous veins of the sphenoid wall and the veins of the carotid plexus are also routes to the cavernous sinus. This is known as the mesial pathway.

4. In fulminating meningitis, which is frequently preceded by an acute pneumococci or streptococci otitis, a blood stream infection would affect the prearachnoid spaces of the cortex. It may also affect the choroid plexus of the ventricles, the ventricles and later the meninges. This is the blood stream pathway.

5. The instrumental pathway caused by perforation into the brain thru the cribriform plate and false passage by frontal probes, needs only to be mentioned.

In 1924, I presented, before the Nose and Throat section of the American Medical Association, the fact that the removal of the middle and superior turbinates in exenteration of the ethmoid cells and opening of the frontal sinuses intranasally, might open the sheaths of the olfactory nerves and thus give a direct pathway to the meninges. It was further stated that the majority of deaths from meningitis following operative precedure in the region was due to this cause. Eighty deaths were cited in the private practice of fifty-two of our leading rhinologists, and in seventy-eight of these, all or part of the middle and superior turbinates were removed.

June 4, 1926, Drs. Turner and Reynolds, of Edinburgh, reported the macro- and microscopic findings in a death from meningitis following an intranasal operation in the region of the olfactory nerves, showing that the infection spread to the brain thru the olfactory nerve sheaths, thus proving my contention of 1924, that there is this pathway thru the sheaths of the olfactory nerves.

6. Paths by Otitic Infection: There are two pathways of infection to the meninges thru the internal ear, one by way of the perilymph, and the other by way of the endolymph.

The perilymph occupies the space between the membranous labyrinth and the bony labyrinth and the two spiral cavities in the cochlea, called the scala tympani and the scala vestibuli. The perilymph is in communication with the subarachnoid space by way of the cochlear aqueduct, which starts on the floor of the scala tympani near the round window and leads thru the petrous bone to the outer edge of the jugular foramen, where the bony cochlear aqueduct ends, but the membranous cochlear aqueduct continues thru the jugular foramen and perforates the dura. Thus there is a direct pathway to the meninges if an infection of the perilymph is present.

The endolymph, which occupies the membranous labyrinth and the scala media of the cochlea, communicates by way of the ductus endolymphaticus thru the petrous bone to the saccus endolymphaticus, which lies within the cranium, or on the external third of the posterior portion of the petrous bone. Ulcerative infections of this space would give direct contact with the dura.

Surgical injury of the semicircular canals, cochlea and

stapes during mastoid operations would infect these two pathways, as would, of course, general suppurative infections of the ear.

7. There are no lymphatics in the brain, and the socalled lymph spaces drain into the large sinuses.

The lymphatics of the head and neck have no communication with the brain, but all drain away from the brain into the lymphatic glands of the neck and then into the portal system.

In the early stages of bacterial infection, the lymphocytes and phagocytes in the lymphatics attack the invader, blocking them at the lymphatic gland. As soon as thrombosis takes place, they cease to function.

This protective drainage by the lymphatics is particularly active in the mucous membrane of the eye, ear, nose and throat, showing the cervical gland tenderness in beginning infections, before the more serious thrombosis takes place.

SUMMARY.

1. PATHS OF INFECTION. Paths left open by embryonic dehiscences, as in the ethmoid bone, the basipharyngeal canal to the sella turcica, and the petrosquamous fissure from the tegmentum tympani.

2. Paths by continuity of diseased soft tissue and osteomyelitis.

3. Venous pathways taken by infections of the face, nasal cavities, mouth and pharynx, causing anterior or posterior thrombosis of the cavernous sinus by retrograde thrombophlebitis.

4. Direct infection by a septic blood stream.

5. Pathways opened by surgical trauma, giving direct communication to the meninges, or injury to the olfactory nerves, thus opening the sheaths that are directly connected with the meningeal spaces.

6. Pathways of otitic infection.

7. The part played by the lymphatics.

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DISCUSSION.

DR. A. H. ANDREWS, Chicago, Ill.: I have been very much interested in this paper. It is more or less unique, in that the essayist selected a subject with well defined limitations, and has stayed well within his limitations, for which he and the society are to be congratulated.

The Doctor makes some statements which I suspect are true, but things I have not thought of before. One was that infection of the cavernous sinus thru the petrosal sinus was more likely to occur if that infection travels up thru the jugular, than downward from the lateral sinus. I never have seen a case where I thought the infection traveled upward from the jugular. It may be that is the reason why we so infrequently have infection of the cavernous sinus in sinus thrombosis.

We have probably all seen serious results come from very minor infections about the nose and face. As the Doctor says, the infection sometimes detours from the ordinary course and goes thru the ophthalmic vein to the cavernous sinus, and meningitis follows. Why it does that in one case and not in another is hard to explain. It cannot be because of the virulency of the infection, for severe infections about the face seem less inclined to this complication than the simpler ones. It may be some special resistance on the part of the patient, and that is a thing that is hard to demonstrate or even to speculate upon. We simply do not know. Why it is that we have necrosis of the walls, with pus in contact with the sinus, without the interior of the sinus being involved, is another matter that is hard to explain, but it is common knowledge, that in mastoid disease, we frequently have erosions of the bony wall of the mastoid, with pus bathing the wall of the lateral sinus over a considerable area, while the sinus itself is not involved. Conditions are different in the roof of the nose. I have an idea, that whenever there is necrosis of the roof of the nose, allowing much infection to reach the cranial cavity, we must have infection of the meninges and serious results.

The Doctor spoke of the avenues of infection from the internal ear. I have never seen and never heard of a case of meningitis in which the endolymph travelled by way of the ductus endolymphaticus. Infection could travel to the cranial cavity thru this channel, but I do not believe that it would be likely to cause a meningitis.

The anatomists tell us that the sheaths of the olfactory nerve follow down the nerve a considerable distance, and that these sheaths are open, varying in different cases, but sometimes as far down as the middle turbinate. This not only allows the cerebrospinal fluid to escape into the nose, but allows infection to pass thru the cribriform plate into the cranial cavity. It is my impression, that a great many times, in operating upon the upper part of the nose, both upon the septum and the lateral wall, these sheaths are opened and a direct channel is produced between the nasal cavity and the cerebral cavity. I do not believe,

however, that infection reaches the brain very often even tho this channel is opened. I think we are much less likely to have intracranial complications when these channels are open, if we do not pack the nose. Packing the nose after operation in this locality holds the infection, and it is more likely to be driven thru these channels, than if we keep packing out of the nose.

DR. JOHN L. MYERS, Kansas City, Mo.: In Kansas City, there has been much interest manifested in this subject for some years. In autopsies, we were finding many cases of cavernous sinus thrombosis. These were in cases having had teeth extracted, or giving histories of having pulled a hair from the nose, or having a furuncle on the upper lip. One case was from infected tonsils, and another from a peritonsillar abscess.

If you will refer to the transactions of 1923, you will find that I made a study of "Tracts Through Which Infection May Be Carried Into the Brain From Extracranial Source." The ptergoid plexus, because of its location (infratemporal and pterygoid fossa), is one of the most common sources thru which infection from the pharynx, teeth, palate, tonsils, muscles of mastication, nose, face, and neck, is carried to the brain. This plexus is connected with the cavernous sinus thru the sphenoidal fissure and the foramen ovale.

Again, it is well to remember that the facial vein has no valves, and in furunculosis of the nares and abscesses of the upper lip, you can see how infection may be carried to the cavernous sinus by this route.

Recently, I saw a case where a pimple above the zygoma was picked with a pin, the patient dying later from the infection. The tract thru which infection was carried to the cavernous sinus was downward to the ptergoid plexus, and then thru the foramen ovale.

I think each of us should familiarize himself with the many tracts thru which infection may be carried within the skull from the external sources other than the ear. In searching the literature, we have found infection has been carried within the cranium to the cavernous sinus, lateral sinus, longitudinal sinus, and pterygoid sinus, thru tracts connecting these vessels with vessels outside of the skull.

I think we ought to report the cases we come in touch with, in order that others may be impressed with the danger of intracranial infection from external causes, and be made more cautious in caring for infected regions outside of the cranium, because of the great danger of intracranial complications thru one of the many avenues which lead within from sources outside of the skull.

DR. RAY K. DAILY, Houston, Tex.: In enumerating the possible channels of otogenous complications, the speaker has not mentioned dehiscences in the roof of the middle ear. Last year we saw a girl, twelve years of age, with a history of an acute otitis media of three days duration. When first seen, at ten o'clock at night, she complained of earache and very severe headache, and she looked very ill. Paracentesis was performed, and there was a free discharge of pus. She became unconscious and developed frank symptoms of meningitis, during the night; the spinal fluid was cloudy, and she died at eight o'clock in the morning, ten hours after we had first seen her, and less than four days after the onset of the disease. Autopsy was refused.

In searching the literature for an explanation of the development of meningitis in the first few days of acute otitis media, we came upon an article by Otto Mayer in the Wiener medizinische Wochenschrift. He reported a similar case with autopsy, gross and histologic findings. In his case, the infection extended from the middle ear to the meninges thru a bony dehiscence in the tegmen; histologic examination revealed the fact that this was not a congenital dehiscence, but one acquired during life. As described by J. Erdheim, these dehiscences are often the result of increased intracranial pressure, which may accompany attacks of migraine, nephritis, or acute infections. This increased pressure has a tendency to cause a bony resorbtion at the base of the skull, and particularly the tegmen. Individuals in whom an increased intracranial pressure has lasted for some time, are left with these bony dehiscences after the intracranial pressure has returned to normal; and for the rest of their lives, these individuals are in danger of developing otogenous complications, particularly meningitis, from an acute otitis media. He advises, therefore, an early mastoidectomy in all cases of acute otitis media, where an increase in intracranial pressure at any time in the patient's life can be determined either from the history or thru a roentgenogram of the skull.

DIFFERENTIATION BETWEEN OPHTHALMIC AND SINUS HEADACHES.

RALPH A. FENTON, M.D.

PORTLAND, OREGON.

Fundamental to the discussion of headache should come brief consideration of the mechanism whereby this symptom becomes manifest. Confusion regarding classification exists in most clinical literature. "Headache" is made to include

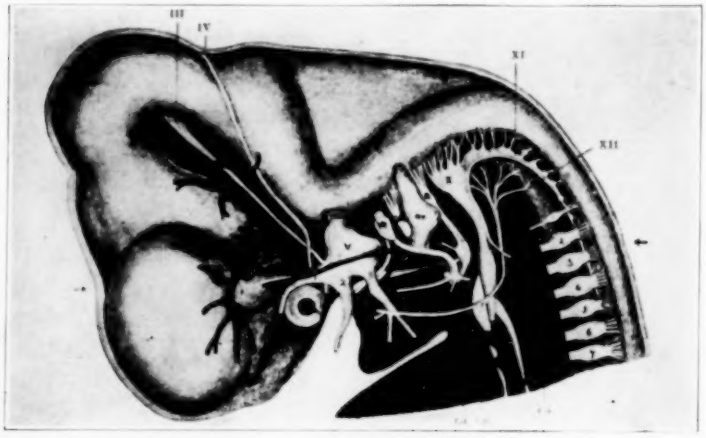

Fig. 1. Distrubution of cerebral nerves and vessels in an embryo of about the fifth week. (Bryce, in Quain's Anatomy.)

both internal ache from changes within the cranium and the meninges, and also neuralgic manifestations, local and referred, external to the brain and its envelopes.[1]

In 1860, Hilton's fourth lecture on "Rest and Pain" contained this suggestive admonition, as valuable nowadays as then:

"If the hidden cause of pain be in any one particular spot, it is only by tracing the nerves of and from that spot that we can hope to arrive logically at the real cause of the symptoms, and so divest the case of its obscurity. Applying this method to practice, it is thru the medium of the distribution of

the cerebrospinal nerves of sensation (the fifth nerve being the true cranial sensation nerve), that we are enabled to explain those pains which are called sympathetic, but which result from a continuity of nerves between the cause and the effect, the disease and the symptoms." [2]

Bryce's reconstruction of a fifth-week embryo (Fig. 1) sufficiently indicates the vast size of the Gasserian ganglion and the simplicity of its central situation with respect to the eye

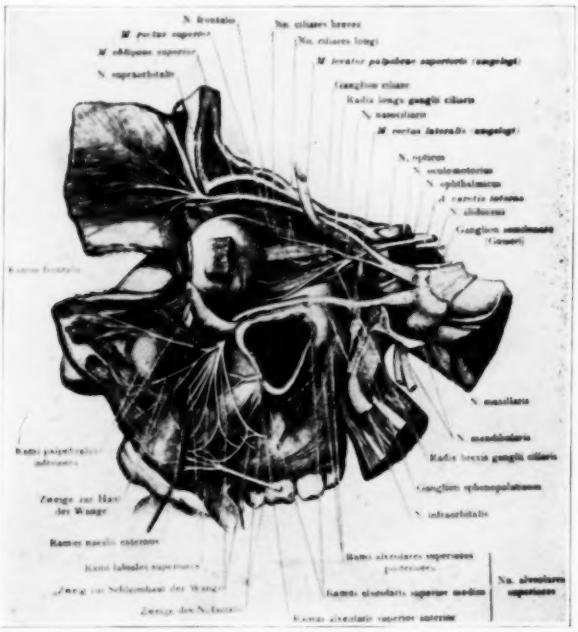

Fig. 2. Distribution of the first and second branches of the fifth nerve. (Toldt.)

and the nose. But with the closure of gill slits, forward development of the cerebral hemispheres, anterior direction of the orbits, and relative arrest of development of the nasal and maxillary system in man, this early embryonal simplicity becomes a maze of complications. (Fig. 2.)

Sensation from the eyes or the sinuses must travel inward over fifth nerve filaments for interpretation or for reference to distant terminals, whose irritation confounds our diagnosis. Pain impulses travel from the Gasserian ganglion into the fifth nucleus, thence to the optic thalamus and the cerebral cortex.[3] Nerves of sensation are found in the dura, but have

so far not been demonstrated in the arachnoid or pia. Regionally, they are about as follows:[4]

Anterior fossa; Nasal branch of ophthalmic division of V. (especially over cribriform plate).

Middle fossa; Extensive branches from Gasserian ganglion of V. and mandibular nerve from V.

(These extend up as far as the superior longitudinal sinus and communicate with the sympathetic plexus about the middle meningeal artery.)

Fig. 3. Cephalic part of the sympathetic nervous system. (Toldt.)

Tentorium; Recurrent nerve of Arnold from ophthalmic division V. near entrance to orbit, running back with trochlearis and ramifying inward to the straight, outward to the lateral sinus.

Posterior fossa; Recurrent branch from ganglion of the vagus IX. root, coming back into dura via jugular foramen.

Sensory branches from the loop between the hypoglossal and the I. and II. cervical to the posterior fossa and occipital diploe.

Vasomotor control and involuntary muscular action is vested in the fibers of the sympathetic system, which travel up from the superior cervical ganglion and along with the carotid plexus (Fig. 3); thence they accompany the cranial nerve

trunks to their nasal and ocular terminations. These fibers traverse the sphenopalatine and ciliary ganglion without actual connection with the sensory fibers in these structures.[5, 6, 7] The assumption that sympathetic fibers carry pain impulses into the cranium and along the dura is not accepted. Nevertheless, such fibers may, if stimulated, readily produce localized changes in the dural circulation, with corresponding pressure and headache. Endocrine function, especially as to the sexual, adrenal, thyroid and pituitary secretions and their

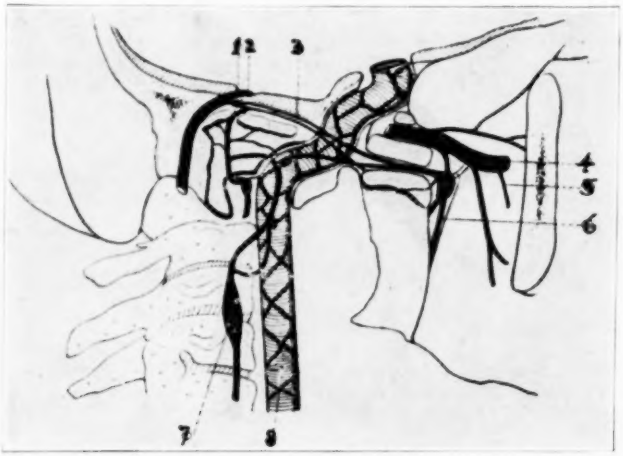

Fig. 4. Relations of the sphenopalatine ganglion with the facial and superior maxillary nerves and the sympathetic system. 1. Geniculate ganglion. 2. Facial nerve. 3. Great superficial petrosal nerve. 4. Superior maxillary nerve. 5. Posterior dental nerves. 6. Sphenopalatine nerve. 7. Cervical sympathetic. 8. Internal carotid artery and sympathetic plexus. (Terracol.)

influence upon the sympathetic and its smooth muscle system, invite consideration here.

The socalled parasympathetic or autonomic fibers, which arise from the VII. and IX. nuclei and travel with the seventh and ninth nerves into the petrous bone, turn off within the pyramid to form the great superficial and great deep petrosals (Fig. 4). These join with a branch of the carotid sympathetic plexus to form the Vidian nerve, running laterally to the sphenoid sinus and into the sphenopalatine ganglion from behind. From this point branches of the superficial petrosal accompany the orbital branch of the maxillary division of the V. to the lacrimal gland; others pass with the sphenopalatine nerve to the nasal mucosa (Fig. 5).[5-8]

Afferent impulses thru the autonomic fibers are carried back thru the Vidian nerve and the geniculate ganglion, and even on down into the cervical and upper thoracic ganglia. Herein we find explanation of referred aches in the ear, occiput and shoulder. These pain impulses are picked up by the posterior root ganglia and relayed on to the optic thalamus and cerebrum, where they are perceived as originating in the ganglionic distribution rather than in the nose or cheek.[3]

The tough dural envelope (Fig. 6) is very sensitive to

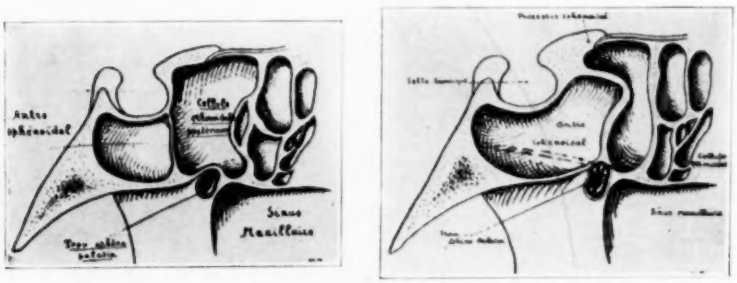

Fig. 5. Relations of the sphenopalatine ganglion area with the posterior ethmoid, the maxillary antrum, and the sphenoid. The course of the Vidian nerve in the lateral wall of the sphenoid is dotted into the right hand figure (usual arrangement). The left hand figure shows no relation between the sphenoid and the sphenopalatine ganglion. (Terracol.)

stretching and pulling. So accurate is the balance of the cerebrospinal fluid within the ventricles and cisternae, that we expect headache when this balance is disturbed by spinal puncture. Thus any change in the specific gravity of the fluid, any increase of contents, whether intravascular or hemorrhagic, cystic, neoplastic, exudative or otherwise, is bound to make pressure upon such branches of the nerves of sensation and vasomotor control as are found in the dura, and thus to produce headache.[9] Toxic substances and proteins (bacterial or specific), gaseous or liquid poisons acting directly or indirectly thru the blood, are probably causative of headache thru sympathetic effect upon the tonus of cerebral blood-vessels, with consequent alterations in the formation of cerebrospinal fluid.[10, 11]

Obviously, interference with free cerebral circulation by degenerative vascular changes, as in arteriosclerosis, angiomata, aneurysms, postencephalitic adhesions, would render such a brain more susceptible to irritations from without.

Alterations in the pressure of the cerebrospinal fluid in the basal cisterna, whether due to change in the fluid or to interference with its circulation, are believed to cause such pressure in the pain transmitting mechanism of the optic thalamus, as to be responsible for the violent disturbances of socalled migraine. This "cerebral glaucoma" may cause lateral pressure on the optic tracts, accounting for coincidental transitory hemianopsia and scintillating scotomata.[12, 13]

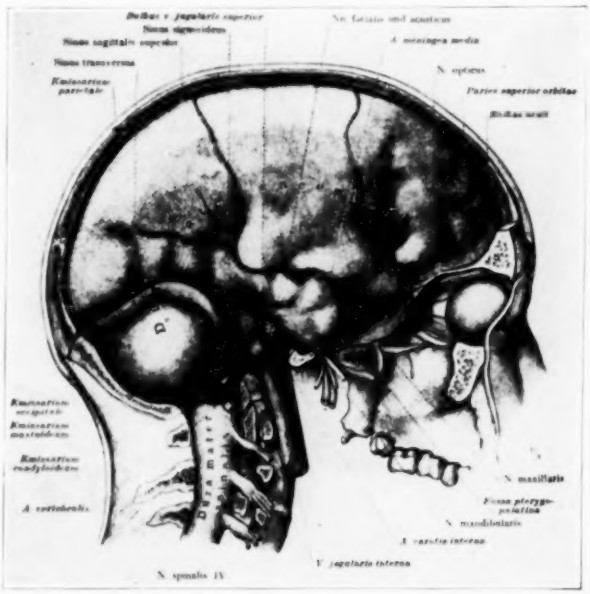

Fig. 6. Lateral view of the dura mater and cerebral venous sinuses in situ. (Toldt.)

Let it be remembered that the sclera is an inelastic case comparable to the cranium, in that pressure upon its contained sensory nerves may readily be caused by changes in the fluid or chemical content of circulatory or lymph channels within the eye. This need not reach the stage of actual glaucomatous blocking of drainage, but may remain in the degree of active or passive ciliary congestion, or even of localized edema or relative hyperemia.

Let it be remembered also, that the erectile tissue of the turbinates passes readily thru every degree of congestion, from pale, waterlogged sogginess to the plethoric and hemorrhagic type.

The network of vessels in the nose and about the eye is accompanied by a far more delicate network of sympathetic nerve fibrils controlling vascular tone. Among these "vegetative" nerves are interspersed end organs of sensation, which report mechanical pressure, edema, hemorrhage, passive or active hyperemia, as well as the results of pressure due to infection and inflammation, whether superficial or exudative.[6-14] Local toxins (bacterial, chemical, resultants of body wastes, fatigue or perverted metabolism) have similar irritant effects upon these sensory terminals.

In general, then, headache from these two fields is representative of local pathology, which is very often actuated thru the sympathetic system, while it is reported to the region where headache is felt thru the fifth nerve and its connections. Perceived within or about the cranium, it may, by reflex stimulation of the sympathetic at the perceiving area, set up local vasomotor disturbances within the dural envelope, which may greatly aggravate the intensity of the original headache.[10] Conversely, conditions within the brain or along the sympathetic or sensory nerves may activate perceptory mechanisms within or about the eye or the sinuses, thus producing irritations or congestions which appear to be local.

With this fragmentary review of the anatomy and physiology of the nervous elements concerned in pain sensation from the eye and the nasal sinuses, we shall attempt to reconcile various clinical pictures reported by authorities in these fields. In extenuation of dogmatic statements which, thru brevity, sound rather too positive, we are reminded of a recent remark by H. G. Wells: "There is nothing so invigorating as a good generalization, but it ought to go thru its facts and marshal them; it ought not to fly over their heads and expect them to follow." *

Sinus headaches, in the opinion of Skillern,[15] are due mainly to pressure, caused, first, by obliteration of the sinus lumen by mucosal swelling, or second, by occlusion of the normal apertures with retention of secretion.[16] Socalled vacuum headache is in his experience uncommon, tho it may be due to similar total occlusion of sinus ostia. Cystic turbinates or high thickenings or deviations of the septum, in so far as they obstruct sinus drainage and promote ethmoidal

*H. G. Wells: The World of William Clissold, N. Y, 1926; v. I, p. 162.

congestion, favor the occlusive type of sinus headache. Bonnier, of Paris, thirty years ago attempted to prevent the sympathetic irritation which he considered causative of nasal headaches by cautery destruction of middle turbinal mucosa over the sphenopalatine ganglion. This foreshadowed Sluder's exhaustive study of sphenoethmoidal neuralgias, which he attributes often to vacuum formation and to ganglion irritation, rather than to the end organ pressure of empyema, or to turbinal and septal malformation.[17, 18, 19]

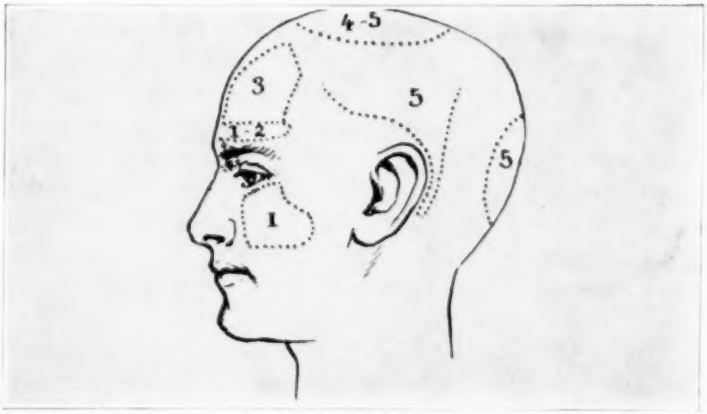

Fig. 7. Schematic illustration of pain areas due to sinus disease. 1. Acute maxillary sinusitis. 2. Acute frontal sinusitis. 3. Chronic frontal sinusitis. 4. Chronic ethmoidal inflammation. 5. Chronic sphenoidal sinusitis. (Skillern.)

Skillern's classical diagram of the locations of sinus headaches is shown herewith (Fig. 7). Frontal sinusitis gives supraorbital to parietal pain; maxillary, gives supraorbital;[20] the anterior ethmoid, at the inner canthus and supraorbital; the posterior ethmoid and sphenoid, parietal, postauricular and occipital.[5-21] Tabulation of the time of appearance of pain and of discharge is as follows; wide variations of course occur:

Sinus	Time of pain	Discharge observed
Frontal	8 a.m. to 1 p.m.	from noon on
Maxillary	noon to night, or constant	on arising and all day
Ant. Ethmoid	constant	on arising and all day
Post. Ethmoid and Sphenoid	paroxysmal, often nocturnal	variable; (lateral pharyngitis)

Excessive local tenderness above the inner canthus, on the thin floor of the frontal sinus, must be distinguished carefully from pain from pressure on the supraorbital nerve in its notch, or from nasociliary neuralgia; but this sign is helpful sometimes in diagnosis of a unilateral frontal sinusitis. It should not be forgotten, also, that many severe sinus involvements will have pain only on the occasion of an acute exacerbation, as following exposure, swimming and the like.[15-23]

Local factors in the consideration of ophthalmic headaches fall into three main groups: disturbances of motility; disturbances of refraction and accommodation; and disturbances of retinal function.

Protagonists of each group have claimed relief from such headaches thru muscle operations and exercises, thru cycloplegia and the correction of ametropia, especially astigmatism of very slight degree, or thru physiologic rest and the use of tinted lenses. Each group has many cures to report; but, as with the undiscriminating rhinologist, many disappointments have followed failure to consider all sides of the given case.[24, 25, 26]

Latent muscular imbalance is frequent, and its increase to manifest proportions is often an occupational hazard. Exophoria, being common, would seem to be frequently causative of headache. Hyperphoria, fortunately rare, almost always is productice of severe headache.[1-27]

Ametropia, whether it be in childhood or age, causes headache whenever use of the eyes is so exacting as to bring about congestion of the ciliary circulation owing to excessive or unequal use of the accommodative mechanism; or, in early presbyopia, owing to an attempt to force inelastic structures into action. Myopes rarely have headache; if present it is due to the astigmatic component of their ametropia, or to other pathologic features of the weakened globe. Irregular pull upon the ciliary muscle in cases of astigmatism, corneal scarring, keratoconus, or posterior synechiae, may often stir up most violent reactions without local manifestations in the eyes.[24, 28, 29]

Excessive demands upon accommodation from tasks at wrong distances must not be forgotten, especially in young hyperopes—factory workers, typists and comptometer operators, and the like.[30] Weakened accommodation from illness,

or specifically from the toxins of septic tonsils or teeth, will activate severe headaches.[1]

Retinal function, if attacked by excessive illumination, as by snowblinding, welding, switchboard flashes and similar intense discharges, is an obvious excitant for headache. Less obvious are the intoxications—alcohol, tobacco, quinin, and others. Definite toxic agents, also working thruout the body, are found in acute or chronic septic states, in arteriosclerosis, in nephritis, in eclampsia, in neurosyphilis, in diabetes, in severe anemias, in socalled "acidosis."[31] Retinal sensitiveness, photophobia, itself an outstanding symptom in such disorders, may lead to headaches presaging the more usual pain of cerebral edema.

Far more common, however, is the dull ache of retinal fatigue, not alone from reading and writing, but in the world of industry. Typesetters, textile workers, machine feeders, gem grinders, glass blowers, dental technicians, armature winders, thread cutters, stereotypers, are but a few of the multitude of specially trained artisans whose eyes may be emmetropic yet causative of headache from actual weariness of the overstimulated visual elements. Obviously general physical tire is a potent factor in such cases. Such patients report "all right after a vacation;" or that "you can stand this job about two years, then you have to quit and do something else."

Acute inflammatory glaucoma is a frequent and most insidious cause of ocular headache, generally unilateral.[51, 52] So frequently is it missed in the early stages, that special consideration must be given to its quiet course and to its simulation of refractive asthenopia, retinal fatigue and the like. Ordinarily, disease of the anterior segment should be fairly obvious: iritis, iridocyclitis, episcleritis and the like. Rarely, an overlooked foreign body, especially if entangled behind the iris or in the ciliary body, will stir up severe headache before local signs become manifest. The dull temporal discomfort and slight photophobia of a beginning sympathetic ophthalmia must not be ignored.

Ocular headaches are commonly frontal,[1-32] altho certain heterophoria and ametropia cases complain bitterly of occipital and nuchal pain,[51] perhaps from impulses relayed thru the sphenopalatine ganglion and Vidian nerve from the muscles and their orbital insertions.

Time of onset depends in general upon use of the eyes,[1-28-29] altho severe fatigue or chronic disease may cause an eye headache to "hold over" and be present on rising in the morning.[29] Intrinsic causes such as glaucoma and iridocyclitis, with their severe orbital and temporal "grinding" pain, are independent of the time factor.[28] The same is true of intracranial pressure conditions, with headache, which show eye symptoms. Thus cysts, neoplasms, gummata, aneurysms, abscesses, fractures, hemorrhage, localized meningitis, hydrocephalus, and other cranial lesions may cause severe headache, accompanied, according to location, by choked discs, visual field disturbances, ocular palsies, nystagmus and other symptoms. Such eye findings are of course secondary to and in no sense causative of the intracranial pressure and corresponding headache.[9]

Congestive ocular pains, especially in acute glaucoma, are frequently accompanied by digestive disturbances and vomiting, so that much time may be wasted in gastric or general treatment before the eyes are suspected.[28, 33, 51]

Physiologic factors affecting our diagnosis include those distant connections of the sympathetic and autonomic systems which may modify the vascular supply of the eye or the nose.[34, 35] Hypothyroid cases, with low basic metabolic rate, are frequently troubled with a nocturnal, occipitoparietal headache which closely resembles the sphenoidal type. This, preceding menstruation, may be an indication of middle turbinate congestion; in fact, spontaneous nose bleed not infrequently occurs in such patients at these times.[36]

A full, throbbing, frontotemporal ache, with facial or bodily signs of giantism, and sliced down form fields above on the temporal side, suggest pituitary involvement rather than the frontal sinuses or sphenoids.

Pelvic congestion of long standing, with corresponding menstrual anomalies, produces headache by interference with vasomotor control rather than by direct interference with the ovarian secretory function. Debilitation from sexual excess will activate existing sinus or ocular headaches, just as any other extreme fatigue can do. No specific endocrine factor is at work in such cases.

Specific substances (foods, pollens, dusts) which cause angiospasm in the eye or nose may do so in the vessels of the dura. Local edema from such anaphylactic reactions may be very painful, or, in certain locations of the brain, may simu-

late very serious lesions of arteriosclerotic or hemorrhagic character. Diagnosis on such headaches is made by vasoconstriction (adrenalin, ephedrin), and by specific avoidance of the suspected protein.[36, 37] An allergic basis for migraine, whether from bacterial toxins from intestinal stasis, or from certain articles of food, seems likely in many cases.

Chronic nephritis, diabetes, intestinal sluggishness and ptosis, cholecystitis, arteriosclerosis, lues, high anemia, leucemia, and numerous other general disorders, must be counted as factors of occasional moment in stirring up ocular or nasal headaches otherwise unimportant. The fundus will often furnish a clue—optic neuritis, retinal hemorrhage, exudates, vitreous opacities, tortuous vessels—in such cases.[29, 33, 38]

Certain individuals are prone to magnify the importance of such a symptom as headache. Morbid psychology and the desire for sympathy may cause complaint long after the original cause of trouble has disappeared. In such cases, fatigue type visual fields and red-green reversal will often confirm the diagnosis of functional rather than organic disease.[1, 39]

A careful history should of course precede any local measures of diagnosis. Often there is a family history of several members with recurrent headaches; this is especially true of heterophoria and ametropia. In the personal history, questions of occupation, use of the eyes, exposure, repeated colds or socalled "catarrh," are quite obvious lines of inquiry. Time of day or night, season of the year, will be indicative of sinus location, of allergic possibilities, or the cumulative effect of use of the eyes. In nocturnal headache, especially with a history of miscarriages, blood and spinal Wassermanns are suggested.

In examination to decide whether headache is ocular or nasal in origin, we must establish physiologic rest—freedom from local irritation or pressure—as a necessary preliminary. If the headache ceases under such investigation, we are on the right trail. Pitres[40] divides the pain from sensory terminals into four levels: 1. Peripheral pain, reached at the sensory ending, as by cocain locally; 2. Pain stopped by anesthesia of the nerve trunk; 3. Pain arising in the root ganglia of the spinal cord, (as in tabes)—this can be reached only by intrathecal anesthesia; and 4. Pain arising from the medulla or cerebral cortex.

We may arrest sympathetic stimulation which is causing local congestion by adrenalin hypodermatically, ephedrin internally, or ephedrin, adrenalin or cocain locally. We may deaden sensory endings in an affected area, thus arresting centripetal pain impulses and their cognate sympathetic stimulations at the more or less distant points to which the pain has been referred. We may place the eyes at rest by holocain and butesin locally; by cycloplegics or miotics; by dark glasses or prisms, and by avoidance of near work and of artificial light.

Application of these methods may be carried out in a rather brief routine. From the history—colds, overuse of eyes or what not—suggestions are likely to arise leading to definite lines of inquiry. Is the headache bad in the morning, better after noon? The frontals come to mind. With a forehead ache, are many handkerchiefs used, and is pus being cleared from the throat? The maxillary antrums should be investigated. Is there a sense of fullness between the eyes? Consider the ethmoids. With pain behind the ear, radiating to occiput and shoulder, we recall the sphenoid and posterior ethmoid. Visual field limitations, especially nasal, with deep boring temporal pain, suggest glaucoma. Bilateral frontal pain, late in the day, suggests astigmatism; if paroxysmal, or complicated by occasional occipital or parietal pain, heterophoria or ametropia may be suspected.[51] Transitory visual field losses, scintillating scotomata with dull parietal aches extending to the vertex, direct our attention to "migraine," with its implications of allergy,[37] of intestinal stasis, and of pressure in the basal cisterna.[12] Browache in middle aged people attempting much close work, suggests increasing presbyopia.

Inspection will show us tired lines and wrinkles in the forehead, and crowsfeet and hammock lines in the lids, pointing to refractive error. Habitual head tilting, sidelong looks, even torticollis, may indicate muscular imbalance. Corneal scars or keratoconus, pigment deposits on the lens, scleral thinning, are surface indications of extensive disease which may be responsible for refractive errors or for referred pain. Ophthalmoscopic inspection will disclose refractive errors, lens opacities, vitreous exudates, retinal edema, hemorrhages, exudates and pigmentation, arteriosclerosis, choking of the discs, or the cupping of glaucoma.

Inspection in the nasal field should always precede the use of washes or shrinking sprays. One may thus detect swollen or cystic turbinates, perhaps impinging above against high deflections which may not impede breathing; septal thickenings equally obstructive of both sides; polypi in the middle meatus; the general direction of purulent discharges.[15, 41] After shrinkage, these items may be pursued further. In the phaynx, lateral inflammation or suprapalatal pus accumulations will be noted.

For relaxation of strain from suspected refractive error, visual fogging by strong convex spheres may be tried. If headache is thus helped, complete refractive measurement under adequate cycloplegia should be undertaken later. The cover test for latent muscular deviations is quickly done, and should never be omitted in any dubious headache case. Frequently rather slight astigmatic errors will give rise to disproportionate pain. If relief be incomplete, trial prisms for near work, or convergence exercises, may solve the difficulty. Often neglected, hyperphoria requires the red glass and Maddox rod for accurate determination. Those suffering from monocular amblyopia are less likely to have headache from muscular and fusion difficulties than are those with fair vision in both eyes.[42, 43]

In the nose, inspection can be completed only after shrinkage by cocain, often with the help of adrenalin or ephedrin. Both middle turbinates should be examined, and cotton should be left in middle meatus long enough to determine the presence of an outflow of pus, if not at first visible. Suction will occasionally cause pus to show itself; it may also give rise to a true vacuum headache, relieved by weak inflation. This may accurately reproduce the symptoms complained of by the patient. Anterior location of pus is usually frontal, but may also involve the anterior ethmoids. Pus in the posterior half of the middle meatus may be posterior ethmoidal, antral, or rarely sphenoidal. Location of the pain and time of the discharge, as heretofore stated, must now be considered. In cases of closed empyema or mucocele, radiographs may be needed. In the posterior cells and sphenoids, the Pfähler or Rhese positions may be necessary. In such cases, lateral pharyngitis and postaural pain may be the only suggestions afforded the diagnostician. Transillumination of the antrum, if done as a routine, is as helpful as the radiograph, and just about as accu-

rate. One is struck with the frequency with which "dark" antra producing frontal aches show no free pus on diagnostic irrigation, altho there may be considerable dissolved mucin in the washings. Recent study shows the frequency of virulent bacterial involvement in these apparently empty antra,[44] whose thickened mucosa may be shown by lipiodol injection and radiographs. Lipiodol may be drawn after the method of Proetz[45] into the posterior ethmoids and sphenoids, to disprove the existence of closed empyema, or to clarify the size and relations of these cavities. Such pictures must be taken immediately after injection. Radiographs will sometimes disclose impacted molars or retained root fragments, which may cause neuralgia very similar to that of sphenoid disease. Dental pictures may not show enough pathology to account for antral invasion of marked virulence; nevertheless, it is wise to view "dead" teeth with suspicion, when frontal pain occurs on the same side. Incidentally, such latent dental infections are a potent cause of low grade iridocyclitis, with headache arising from difficulty in managing the inflamed accommodative mechanism.

Relief of congestion by bodily rest will commonly help both types of headache. Lying down quiets the circulation, and is often the only relief these people acknowledge. Jugular compression by tight collars, or venous stasis in the head from bending over, increase these headaches. Cold packs across the eyes will frequently allay the pain of refractive or muscular weakness or spasm, while that of frontal involvement may be rendered more violent. Hot packs may aggravate the pain of congestive ocular lesions, while affording some relief, apparently from transudation thru the turbinates, in acute sinus disease. Many are similarly relieved by steam inhalations or hot saline irrigations. These findings are however most variable.

Relief of intracranial tension with corresponding headache is secured by strong saline purges, or even by strong saline injections intravenously. This forced dehydration secures immediate reduction of intracranial pressure, but it is a therapeutic rather than a diagnostic measure.[9, 36]

Locally employed agents for decongestion include adrenalin, ephedrin and cocain. In accordance with the history and the local condition, we may shrink swollen tissues and do away with nerve pressure; then anesthetize the irritated areas and

note the effect upon the distant pain. This is especially effective in the region of Meckel's ganglion. The period of relief following local use of dionin in the eye, after the first hour or two of severe chemosis, may possibly be a morphin effect; but to us it has seemed rather a decongestive effect by transudation.

In conclusion and recapitulation of these crowded, elementary observations, we are reminded of:

The history, not forgetting
family tendency,
recurrence,
exposure, (weather, occupation, irritants)
amount and kind of use of eyes.

Based on the history, we look for
Nasal obstructions to drainage:
injuries; malformations;
septal and/or turbinal;
inflammations,
edema (allergic or toxic),
neoplasms.

Ocular interferences with circulation and
lymphatic drainage: (corneal, iridociliary),
inflammations, (foreign bodies),
increase of tension,
edema (allergic or toxic),
changes in the media,
changes in the retina and nerves;

Muscular insufficiencies;

Refractive and accommodative errors.

The particular group of nerves which is irritated must be sorted out and set at rest, thus stopping or diminishing the headache, at least temporarily.

Such rest may be effected by:
local ischemia and/or anesthesia,
avoidance of specific irritants,
avoidance of irritating tasks.

The headache may be central in origin, with symptoms referred to the eye or nose: relieved by general, not local measures; eye or nose symptoms are diagnostic of the general ailment: cerebrospinal, cardiovascular, gastrointestinal, renal.

The headache may be psychic, with symptoms referred to the eye or nose: fatigue phenomena and unstable, irregular symptoms will be manifest.

We must not forget that ocular and sinus headaches may exist at the same time in the same patient.[38, 46, 47, 48, 53] Nor should we forget the important role played by degenerative general disorders in building small amounts of ocular deterioration or nasal stasis up into relatively serious complications.

No task in diagnosis is more difficult than the evaluation of all factors in these complex cases. Nowhere in medicine is an accurate history more valuable. With some searching of conscience, we may recall that S. Weir Mitchell, master healer of psychic pain, urged us over fifty years ago to beware of the easy alternative in diagnosis—hysteria or neurasthenia—and to look more carefully into the details of our own special fields.[49, 50] Differential diagnosis can never rest upon headache alone.[52, 53, 54]

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DISCUSSION.

DR. WALTER R. PARKER, Detroit, Mich.: Dr. Fenton has given us a very exhaustive anatomic review of the possibilities of the location of lesions that may have headache as one of their clinical manifestations. I agree with Dr. Fenton entirely in his closing remark, when he says that headache alone is rarely sufficient to make a diagnosis. It is perfectly true that certain lesions cause definite pains in well defined regions, but unfortunately, no one lesion has a monopoly of any area.

There was one statement made by Dr. Fenton that was not in itself misleading, but might lend itself to misinterpretation. He speaks of glaucoma as a frequent cause of pain. Acute glaucoma is always associated with pain; simple, noninflammatory glaucoma almost never. And as the simple group is much more frequently seen than the acute form, it would be a little misleading if we were to classify glaucoma as a frequent cause of head pain. If we relied on pain in the diagnosis of simple glaucoma, I think we would fail to recognize the disease in the vast majority of cases.

Not infrequently we fail to cure headaches thru the treatment of the eye or the extrinsic muscles, when as far as the history is concerned, the case is typically one of ocular headache. We are somewhat consoled, however, by the fact that the use of glasses occasionally cures headaches which have not been relieved by one or more operations on the nose or sinuses. As de Schweinitz has said, "Pain is an artful deceiver."

We are sometimes asked by family physicians if their patients could have enough refractive error to be responsible for headaches. As a matter of fact, there is no direct relation between the amount of the refractive error and the severity of headache; a small error may be responsible for severe headache. The moment the refractive error is so great that it cannot be overcome by accommodative effort, the patient sees less but is comfortable.

We have all had cases that gave a history of headache until the onset of presbyopia, and occasionally a patient who has had his headaches relieved by the use of distant glasses may lay them aside with perfect comfort after reading glasses are required.

An accurate knowledge of the possibilities of pain in these complicated cases cannot be overestimated, and that is the great thing to my mind that Dr. Fenton has given us. If the lesion is definitely located, this accurate knowledge will explain why the pain is in a certain area.

DR. HARRIS P. MOSHER, Boston, Mass.: I wish the writer, in attempting to be complete, had not been so complete, because the effect of the paper to my mind is quite disastrous. I wish he had given us his personal experience. But I feel the subject is of tremendous importance. It seems to me that to evaluate headache from any standpoint, you need all the horse sense you can muster, and all the general medicine you can muster or get from your consultant. That is practically all I have to say about headaches, except to ask a few pertinent questions.

First, in his experience in treating the eye, ear, nose and throat, what has been the predominant cause of headache? Will he kindly put himself on record? As a nose and throat man, I find the sinuses, frontal, then ethmoid, and finally the antrum.

Second, how many cases of Meckel's ganglion headache has he seen, how many has he cured, and how has he cured them?

Third, what does he believe on the question of headaches due to nasal contact back between the middle turbinate and the septum? How many cases has he seen referable to that area, and how many has he cured?

Finally, how many cases of vacuum headache has he seen and cured? And last of all, will he please tell me for my good, and I hope for the good of some of the rest of you, what is the chief cause of headache in his experience as an eye, ear, nose and throat man?

Dr. R. H. T. Mann, Texarkana, Tex.: Viewing my own practice in ophthalmology and otolaryngology, I think that every case of headache should be examined, both from the standpoint of the otolaryngolgist, and from that of the oculist. I think that very many of these headaches will be found to be due, not to a single cause, but to several causes. I do not think it is just to a patient, who is depending upon the use of his eyes for a living, to test the muscles, correct the error of refraction, and expect that patient to be relieved from headaches. I think the patient deserves and needs not only ocular correction, but the correction of all existing troubles of the nose, throat and tonsils as well. Just one case to illustrate what I am trying to say: A young student had given up the idea of studying medicine, because his excessive headaches had not been relieved by the use of glasses. There was a slight change made in the glasses, his tonsils were removed, and he could read all night.

Dr. Ross H. Skillern, Philadelphia, Pa.: I will speak from the sinus point of view, and give you my more or less slight experience with it.

The cause of headache in sinus conditions, according to my experience, is due to several things. First of all, to the direct irritation of the nerve ends in the sinus mucosa. We know, that when the sinus mucosa becomes swollen and edematous from round cell infiltration, it is hypersensitive. We know, that when we go into the antrum under local anesthesia, we can have perfect anesthesia in the canine fossa, and it will continue perfect until we get to the antral mucosa, and then there is pain until we have made a slight opening and packed it with gauze saturated with a strong solution of cocain. I think the vacuum headache has very little to do with it. At first, we accepted the vacuum theory, and I was quite sure of it when we took off the end of the middle turbinate with immediate relief. But how about the middle turbinate being the cause of headache rather than a vacuum above? It has been my experience that aeration is the great factor in relieving a sinus headache, and not drainage. We have seen cases from time to time where we got practically perfect drainage, but the aeration was not quite sufficient, and in these the headache continued, whereas in other cases, where we had thoro ventilation, the headache disappeared. So I believe that ventilation is the crux of the entire situation.

Speaking from that point of view, I think the headache is due first of all to a swelling and irritation of the mucosa—there may be a slight vacuum—but more directly to the irritation of the mucosa itself, or to a swollen middle turbinate jammed in there and causing increased conditions of pressure on that side, the headache continuing until the condition of pressure is relieved.

Dr. Ralph A. Fenton, Portland, Ore. (closing): I am extremely grateful to these distinguished gentlemen for their willingness to undertake a discussion of this subject, because their pronouncements are authoritative. I am especially glad to have Dr. Parker's classification of pain in glaucoma.

Of course the trouble with this whole thing is that both sorts of headache travel down the same nerve pathways, so we must look at the entire clinical background to find the cause of the condition.

As to Dr. Mosher's questions, assuming that my practice happened to be equally divided at any given time, my guess would be that perhaps headaches of nasal origin might be 40 per cent., and those from the eyes 60 per cent. Other people would feel differently. I think that would vary largely according to the geographic location and the class of people who happened to be coming. If you are dealing with a large number of industrial cases, you would have more industrial injury cases with headache. The same might be said of one type of climate and another. Such questions in the nature of things must be reports on a limited number of cases. I have attempted to justify accepted opinions regarding headache as a diagnostic factor. In a rather limited way, the paper reflects our feeling about the relative importance of the various sinuses as the cause of headache. Certainly they are an outstanding factor.

Regarding Meckel's ganglion, contact points, and vacuum headache, I think that in all these conditions we are prone to lose sight of the fact that Meckel's ganglion is really a switchboard on the way back from the nerve endings to the Gasserian ganglion; diseases of Meckel's ganglion, itself, are very rare. The disease is at some place along the nerve endings, or around the ganglion, and pressure in that neighborhood would have the same effect as if you were to throw the switch in the back part of this room—you would put out the lights. There would be no trouble with the lights, but simply an interruption of the current. This is an essential factor in most of the work done regarding Meckel's ganglion.

So far as vacuum headache is concerned, I will frankly say that in a small way we can corroborate what Dr. Skillern has said—we do not see vacuum headaches. We can cause them by the use of suction in the nose, and then by inflation we can give immediate relief. But I do not think this is as important a clinical factor as we thought in the past.

SINUSITIS FROM THE VIEWPOINT OF AN OSTEOMYELITIS.

G. B. Trible, M.D., F.A.C.S.

WASHINGTON, DISTRICT OF COLUMBIA.

It is hard, often, to put into words, or to give definite expression of ideas which may have been long prevalent in the minds of many men. All of us have felt that operative procedures on the sinuses have shown unfavorable results in instances not explainable either by anatomic malformation, or by the virulence of the infective agents; in fact, the apparently mild infections with classic anatomy have often proved to be the most intractable to treatment. There is a vague discontent, an innate dissatisfaction in our minds, a groping for a better understanding of the problems presented, and an intense yearning for a better outcome. For this reason, your attention is invited to a phase of the question which seems to be coming more and more to the front. If it be remembered, that for the most part, all the cell bearing bones of the head are fundamentally flat bones, the inflammatory changes that take place must be somewhat analogous to such changes in the flat bones in other parts of the body. A consideration of the infections involving the sinuses, from this standpoint, makes their seeming vagaries more rational and clears up for us some of the problems with which we have to deal.

In the tabular bones of the head, the osseous tissue is laid down in the mesenchyme, an embryonic connective tissue lying between the brain and the skin. Where the later bone is to form, the mesenchyme becomes more richly vascularized and active proliferation of the cells takes place, the characteristic osteoblasts are found and are grouped off in the hyalin groundwork, and become the first bone cells; then there is a later deposit of bone salts. This bone is spongy, and is resorbed and deposited simultaneously to permit expansion of the brain. This destruction is carried out by large, multinucleated cells, the osteoclasts, which appear closely applied to the inner surfaces of the trabeculae. The origin and the

morphologic characteristics of these cells are subject to some dispute, but their constant presence cannot be gainsaid. This hollowing out of the bones of the skull, which later bear air cells, is a continuous and a perfectly normal reaction, going on till death, as we note especially in the antrum. There is a certain balance in this process, and when we find it grossly interfered with, as illustrated in suppurative conditions of the antrum in childhood, not only that sinus, but the corresponding frontal air cells develop but poorly.

The development and structure of bones is roughly the same, whether long bones or flat bones, each consisting of a dense outer cortex, surrounded by a highly vascular membrance, the periosteum, and having on the inside a more spongy, diploetic layer, more richly supplied with vessels than the outer, denser layer. In long bones, there is an actual channel, filled with marrow, blending into the cancellous bone at the joints. Pathologic bone changes will then be somewhat different in the flat bones, and cannot be viewed in the same light as bone changes in the long bones. For a practical application, when we come to the sinuses, experience teaches us that pathologic changes in the mucosa and the mucoperiosteum will not explain all the failures, nor will the establishment of aeration and drainage render a success certain. The consideration of the deeper histologic changes will show that these cases are those of osteitis, or an osteomyelitis, and the surgical impracticability of removing all the invaded structures explains the inability to reach success, from purely surgical procedures, and will explain the occasional sudden violent extensions after operations. An analogous situation in the other flat bones, such as the ilium or scapula, is sometimes found. These bones, being simply long thin rows of cancellous tissue between compact layers of cortex, will show the same stubbornnes and tenacity in resisting surgical treatment. Different terms are applied to inflammation of the bones, depending upon the site, such as periostitis, if there be involved only, or chiefly, the periosteum; osteitis, in the superficial layers of the cortex; myelitis, when only the medullary space is affected; osteomyelitis, extending deeper thru the cortex and involving the medullary space, tho one may lead to another and all be present at some time during an attack, or a combination may exist.

We find, further, that osteomyelitis can be subdivided

into localized or circumscribed varieties, with very little tendency to spread, and which will heal after the focus is removed, or free drainage has been established and the reparative processes commenced; and diffuse or spreading osteomyelitis, in which spontaneous healing does not take place and the tendency is to spread, in spite of all measures undertaken. Portals of infection in either long or flat bones are: first, blood or lymph, directly thru to the medullary structure—not so frequent in flat, as in long bones; second, by an origin beneath the periosteum; third, by direct inoculation by trauma, and in this connection would appear those cases of operative origin.

The radiographic changes in bone involvement are not so characteristic in flat bones as in the long. Infection spreads along the Haversian canals; the calcium salts lying between the canals are relatively slowly absorbed; infection travels, leaving islands of bone between the areas of infection. If they completely surround a piece and it is cut off, a sequestrum forms, much rarer in flat bones than in long ones. Beginning changes are not readily shown in radiograph, while persistent shadows are due to alteration in the structure, or deposits of calcium salts in the periosteum, and may continue after symptoms have subsided, but fluid or secretions, or even polypi, do not cause such marked shadows. In cartilage, the infection always starts from the periphery, never from the center of the mass. These processes do not occur at random but follow well defined laws. If destruction is not much more rapid than regeneration, we have little disturbance of the body economy. An example of slow bone changes can be borrowed from dentistry, and is made use of by orthodontists. They move teeth in young maxillae, depending upon pressure to rarify the compressed bone, which then is absorbed by the osteoclasts and the carbonic acid content of the blood, and new deposits back of the teeth to fill in the spaces are made by the osteoblasts, the action going on simultaneously.

In the long bones, such as the shaft of tibia, treatment of all osteomyelitic invasions has become practically standardized. The primary bone changes, such as lues and tuberculosis, are relatively infrequent in surgical conditions involving the accessory sinuses, tho of course common in the long bones. Malignancies of the bones around the sinuses, or in them, are harder to differentiate than elsewhere by radio-

graphic examination, for the characteristic concentric growth can not be made out. The modification of the periosteum, in the field with which we have to deal, undoubtedly influences to a great extent the conditions. There is no true periosteum in the lining of the sinuses, with the possible exception of the sphenoid; simply the layers of the mucous membrane modified, a mucoperiosteum. This mucoperiosteum is intimately connected with the ciliated columnar epithelial lining of the cavities, and is subject to more possibilities of direct infection than the periosteum in other portions of the body.

When osteomyelitis is mentioned, our mental picture is due to its association in our mind with the long bones, richly supplied with marrow, subject to massive destructive changes; or if it be not that, we think of the great extensive necroses typified in the frontal. Further consideration will bring up that in children with separated, not yet perfectly united bones of the skull, this last is rare, while with adults, with closed suture lines, gross destruction is found. This is in direct contrast to the children, who show more frequent rapid bone invasions in long bones, rarely in the walls of the sinuses, except in the upper maxilla, which Blair believes in some instances to be due to antrum or nasal infection. Our conception must not be based on these severe cases, for fortunately such are rare, but by their very intensity have made such an impression, that the minor, more chronic changes have been overlooked. Gerber, quoted by Hajek, has made a rather complete study and reported but comparatively few, and many of these postoperative. They are few, somewhat from the same anatomic and histologic reasons that the type to be described is common, a seeming paradox, but understandable when the brief histologic review is considered. Given a sinus, with thin mucosa, the deeper layers taking the part of the periosteum, subjected from infancy to repeated mild infections, having a normal tendency thruout life to the resorption and deposit of bone, delicately balanced as is Nature's handiwork always, and in the case of the maxillary subjected to a double attack, both from above and from below—the stress of mastication, the infections around and about the teeth—the wonder is, not that there is so much, but so little damage, and that the natural reparative processes have been able to act as efficiently as they do.

The classic picture, then, of an osteomyelitis, is that of a

disease, inflammatory in origin, involving the bones and having its point of inception in the medullary tissue, tho at times arising from the periosteum. In the cases carried thru the blood stream, infection may be directly beneath the periosteum. In the other types, the infection may go in with the nutrient arteries and involve the medullary space directly. In later adult life, the resorption of the diploe and the deposit of cortex modifies the picture, and it is sometimes seen as minute bone abscesses, resembling a rarefying osteitis. The acute stage of an osteomyelitis may be absent, or may clear up, with the formation in the long bones of a fistulous tract and spontaneous drainage, or with surgical intervention, the condition may become quiescent. When it is recalled, that in a sinus, drainage can be easily established, the infection for the most part entering thru a superficial necrosis directly under an eroded area of the mucoperiosteum, or by an island of infection around a nutrient or perforating vessel, or by an extension of a thrombophlebitis, it can be seen that there is no deep structure to be penetrated for the pus to escape, and it would not follow that there would be a marked rise of temperature, or marked leucocytosis, such as would be encountered in a similar invasion of a long bone.

The different sinuses have varying powers of resistance; as before mentioned, they all have in common a feature of a loosely attached mucoperiosteum. In the frontals, the margins are extremely susceptible, and in addition, the walls of the nasofrontal ducts are often involved. In this case, it is probably because this latter area is the most dependent, that secretions tend to accumulate, the mucosa becomes edematous and breaks down. Pus penetrates, softens and erodes the periosteum, directly attacks the superficial layers of the bone, and we have established a definite bone infection. In the case of the sphenoid, the lining membrane more closely approaches true periosteum, but we have here also a deep recess beneath the point of drainage, and the same accumulation and decomposition of secretions, with invasions of the floor and walls.

In the ethmoid, a different condition prevails; partly from its innate embryologic and histologic structure, there is less tendency to a direct necrosis, but more frequently there is the production of a low grade inflammation, which has been studied in great detail by McMahon, tho studied first by Sluder. In this connection, too great credit cannot be given to

the German School abroad and to the work of Skillern, Sluder, Mosher, and Beck in this country, as well as others who have endeavored to place this branch of surgery in a sound position. McMahon's conclusions were, that there were found five marked changes in the bone; rarefaction, necrosis, osteomalacia, fibrosis and hyperostosis, and he felt that the constant changes in the walls of the blood vessels, diminishing the supply of blood to the part, caused the production of connective tissue. As might be expected, the changes in the ethmoid and middle turbinate partook of the same characteristics.

In the case of the antrum, we find a great complication by extension of disease from the teeth, granulomas, softening and rarefying the bone or pushing up the periosteum; abscesses at the apices, bursting into the antrum, or simple extension, or thru thrombophlebitis. The mere fact that the upper jaw in the antral area is adentulous, does not exclude that as a possible source of infection, particularly in the low grade cases. The sockets from which infected, pulpless teeth have been removed, unless curetted and cleared out and healed from the bottom, tend to close over, become occluded by cellular proliferation, the infection is locked in, and the same environment for anaerobes established as existed previously. A continuation of this process will affect the antral floor, either directly or by extension from a thrombophlebitis. Glandular deficiencies, or metabolic abnormalities, especially of calcium, are contributory, tho up to the present, we have been unable to find characteristic and constant blood changes. There is undoubtedly a certain association or mutability of related conditions, such as asthma, hayfever, and vasomotor rhinitis, tho the after results of these conditions is not usually a bone change of the type of an osteomyelitis, but more frequently an osteoporosis, an absorption of bone salts and a rarefaction of the bone, probably due to the unregulated activities of the osteoclasts.

In analogous conditions involving the accessory cells in the temporal bone, the tendency is to the overproduction and deposit of bone, as in chronic otitis media or mastoiditis, an osteosclerosis rather than an osteoporosis. There is less tendency for an unregulated absorption of bone in the temporal than in the other air cell bearing bones of the skull.

A gross illustration of an acute osteomyelitic process will be found in orbital abscess, in the erosion of bone into the orbit, or thru the skin and discharge externally, from the ethmoid or frontal. Similar softened osteomyelitic areas may go thru the inner plate of the frontal, and give rise to extradural frontal abscess or frontal lobe abscess. The bacterial agents are not uniform, tho they are more frequently staphylococci. The more violent infections from streptococci run a more rapid course. In the cases of dental origin, the sewage streptococci, which are facultative anaerobes, are often found, and the pus is characterized by a fecal odor.

The question naturally arises: How can this situation be met? The acute sinus cases, not especially virulent, are simply a continuation or an extension of an inflammatory condition of the nasal mucosa, and subside with its quiescence. The more virulent, if in danger of erosions and involvement of deeper structures, need thoro intranasal drainage and aeration. The fulminating, with prostration and danger of meningeal or cerebral involvements, will likely require external operative measures. The low grade ethmoidosphenoidal cases, as emphasized by Sluder and his school, are probably best handled according to his methods. It is when we come to the type of cases, chronic in point of time, showing evidence of erosions of mucosa, polypoid degeneration, or involvement of the bone, that the use of all remedial measures is a necessity. Glandular therapy combined with calcium, ultraviolet light exposures, diathermy, attention to diet, suitable change of climate, conservative but persistent attention along surgical lines, must all be called into play.

DISCUSSION.

DR. ROBERT SONNENSCHEIN, Chicago, Ill.: I wish to express my appreciation of this paper. While it is true that his research work is of the highest order, I am reminded of a statement made by Dr. Norval Pierce a year ago—when a member presented a paper both scientific and practical—that he thought the ideal paper was one which gave the results of both scientific investigation and clinical work. We have an example of that in Dr. Maurer's article, which I think is excellent.

The quotation by Dr. Arbuckle of Mackenzie's statement, that often a minor procedure upon the sinuses is followed by extensive osteomyelitis, brings to mind a case that came to us about four months ago. A young woman, about 20 years of age, had had a subacute maxillary sinus infection for some months. After repeated irrigations failed to relieve her, under local anesthesia, we made a large opening into each maxillary sinus and inserted a rubber tube. On the right side, the result was fine; on the left side, the patient developed in about ten days very severe pain in the cheek for which we could find no explanation. She developed swelling in the lacrimal region, a fistula formed, and just about that time she passed out of our hands. I learned some weeks later, that the patient was critically ill with a diagnosis of frontal lobe abscess, and she died of a meningitis a few days later. Unfortunately, we have been unable to obtain the details of the last days of her illness. I do not know whether postmortem was held, but the case emphasizes what Dr. Arbuckle has said, that from a minor operation such as I have outlined very serious results may ensue.

PARANASAL SINUSITIS, A CLINICAL CONSIDERATION

JOHN J. SHEA, M.D.

MEMPHIS, TENNESSEE.

In these days of scientific medicine, we are inclined to rely upon the laboratory for our information and to neglect the clinical study of our cases. If we study our own patients and observe their clinical course, we will improve our diagnostic acuity and render to our practice better service. It is not the intention of this paper to cover the entire subject of paranasal sinusitis, but to limit our remarks to certain phases of the subject, some of which are accepted facts, and others clinical observations.

ETIOLOGY.

HEREDITY: A tendency towards sinus disease is often the fruit of the family tree. The frequency with which the children of parents suffering from sinus disease contract the disease early in life, is a well known fact, and irrespective of environment, diet or possible contagion, I believe that an inherited weakness towards the condition is a most important factor. This inheritance may be either direct or crossed, the latter being the more common. The sinuses of the child assume the same morphology as those of the parent or immediate antecedent whom it resembles in facial appearance, if the child and the parent have normally developed sinuses. This knowledge can be used to determine whether or not the child's sinuses are developing properly. We have rayed a number of parents with persistent infantile sinuses, and have been unable to show that their offsprings have a prototype of these dwarfed sinuses, and in our series, we have had children with persistent infantile sinuses resembling in facial appearance a parent with a mature set of sinuses.

BACTERIAL VIRULENCE AND SELECTIVE ACTION: This varies in each epidemic of upper respiratory infections. Likewise,

the tendency towards sinusitis varies in the different epidemics, and at times the inflammation tends towards the formation of empyema.

THE INFLUENCE OF THE REMOVAL OF THE TONSILS AND ADENOIDS IN YOUNG CHILDREN: This is a subject worthy of the consideration of a special study, and after careful observation, it is our opinion that children who have suffered the loss of their tonsils and adenoid at an early age, are more inclined to succumb to sinus disease than children who have retained theirs until puberty. The logical explanation of this is, that the sinuses play some part in the development of immunity in upper respiratory infections. The secretion found in an acute nose during the early stage of an inflammation is not only a physical protection to the membranes, but has antibactericidal powers. When the tonsils and adenoid are in situ, they aid in producing antibodies, and after their removal, the sinuses are called upon to supply this deficiency. The ability of the sinuses is apparently limited, and in time they fall prey to the infection they are combatting. In the study of fifty-two children below the puberty age, who sought relief from symptoms suggestive of sinus disease, I found that thirty-five had undergone operations for the removal of their tonsils and adenoid. Thirty-three of this number gave the history, that their symptoms referable to the sinuses appeared after the removal of their tonsils and adenoid. The earlier their tonsils and adenoid had been removed, the more susceptible did they seem to sinus disease. Eight children in this series had lost their tonsils and adenoid and contracted sinus disease before they had reached six years of age. I had removed the tonsils and adenoid of the majority, and personally observed the subsequent onset of the symptoms and signs of sinus disease in these children. In contrast, of seventeen children who had retained their tonsils and adenoid to their tenth year, only eight had developed sinus disease. My attention was first attracted to this phenomenon by parents returning with the complaint, that since their children had been operated upon for diseased tonsils and adenoid, with each subsequent cold, the children would suffer nasal inflammations and would clear their throats and cough. The period between the operation and the onset of the nasal symptoms is as a rule two winters. During the first winter, these children enjoy good health and are free from disturbing infections, and it is my belief that during this

period the sinuses are capably manufacturing sufficient antibodies, but their ability being limited, the sinuses readily fail to carry on. The tonsils, being inhibited by the gonads after puberty, are not thereafter such a power in autoimmunity, and their absence does not enter into the clinical picture. It is not my intention to discredit tonsillectomy during childhood, for I appreciate the wonderful results obtained in the majority of cases.

Pathology.

Discharge: The character of the discharge as observed after lavage will aid in determining the stage of the inflammation. When the discharge begins to agglutinate, the stage of immunity is being approached. This fact further substantiates the theory that the sinuses are active in the process of immunity of the upper respiratory infections.

The Mechanism of the Development of Inflammation in the Sinuses: This may be primary or by extension, the latter being observed when an empyema of one of the sinuses is the terminal stage of an acute infection of the upper respiratory tract; the former, when the first reaction to the infection begins within the sinuses.

Chronicity: Each succeeding acute infection interferes with the nutrition of the membrane of the sinuses and their bony framework, and thru faulty metabolism, the process of repair is weakened. Seldom does the sinus become chronic from the initial infection, even tho mild. Normally developed sinuses are less apt to become chronic than the persistent infantile type, because the latter have thru infection during their formative period been arrested in their growth. The resulting hypertrophies of the nasal membrane and turbinates, and the growth of polyps from recurrent infections, by blocking the ventilation of the sinuses, keep them in a state of chronic inflammation. Eventually the bony framework of the sinuses becomes chronic thru faulty nourishment.

Vertigo: This is a common symptom of acute frontal sinusitis, and rather constant whenever the patient stoops over and straightens up into the erect position, but its importance as a symptom in chronic frontal sinusitis has been neglected. During the past year, two patients who had been referred for the Barany tests and an examination of the nose and throat showed in their roentgenograms shadows suggestive of osteoma within the frontal sinus, and a third referred

for the same examinations showed a dental cyst which, when pressed upon, would excite an active vertigo. The latter phenomenon could not be explained. While experimenting with negative pressure in the sphenoidal sinus, I found that several of the patients at times would exclaim that they were having the same sensation as felt while diving. Positive pressure within these sphenoidal sinuses failed to influence equilibrium.

Sugar Tolerance: (Fig. 1.) If a normal individual is

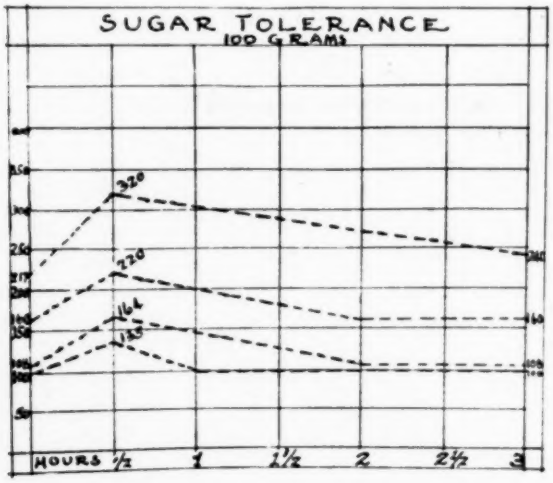

Fig. 1. The lowest line was a test of a controlled case, and the sugar rose to a concentration less than the normal. The second line is that of a mild case of arthritis in which no focus of infection could be demonstrated. The three and four are of the same diabetic, the fourth being made during an attack of maxillary sinusitis.

given 100 grams of sugar, his blood will show in a half an hour its greatest concentration, which is as a rule 0.150, and by another hour will have reached the so called normal of 0.100; but if the same amount of glucose is given a sinus patient, it will pass the concentration of 0.150, and will require more than an hour to return to its basic level. If a diabetic patient develops a suppurative sinusitis, he will show a greater intolerance toward the glucose, and in one of our diabetic patients with an acute frontal sinusitis, his blood sugar rose to 0.700 on what we supposed was a low sugar diet. With this knowledge that sinus patients have a low sugar tolerance, it is my practice to remove glucose from their

diet, and as they improve, to allow of other "sweets" than sugar. Honey is the best tolerated of the "sweets."

Diagnosis.

Roentgenology: It has been stated before national societies that the knowledge obtained from roentgenograms of the sinuses is unreliable and of little value. This is the result of poor cooperation between the rhinologist and the roentgenologist. No set rule of timing can be followed in the taking of sinus roentgenograms, and often several sets of pictures are necessary to obtain the best results. We are taking today five exposures for the routine examination: (a) The "23° angle" by the Law technic, which gives the surgical relations of the ethmoids, frontals and antra; (b) The "chin-nose" or Water's position, which demonstrates the floor of the antrum; (c) A lateral with the right side down, centered thru the posterior ethmoidal cells, (d) The same with the left side down; and (d) a vertical thru the vertex, which gives the best view of the sphenoids and the posterior ethmoidal cells. The latter view also confirms the findings in the antra. In the past, sinus pictures were made with too powerful penetration, and the result was a roentgenogram of the skull, and all of the sinus pathology was wiped out of the picture. In order to obtain the most knowledge, one or more of the views often are taken over, with more or less penetration as the case may demand. If the pathology is limited and within the sinuses, the film should be soft, and if the changes sought are in the bony framework, the film should be harder, as for skull roentgenograms. In the examination of suspected malignancies, the series should have at least three degrees of penetration, soft, average and hard. The first step in the interpretation of the roentgenogram is the determination of the stage of the development of the sinuses, for the sinuses reflect in their growth and development the infections they have suffered during their formative period. Fortified with this knowledge of their past history, the morphology of the right sinuses is compared with that of the left side. If in the Law view the orbital shade is used as a standard for comparison, it should be slightly darker than the ethmoidal or frontal sinus shadow. The latter two should be of the same shade. The roentgenogram that is most often misread is that of the cystic type of ethmoids, for in this picture, the ethmoids appear clear, in

fact they are too clear, and signify that the membrane and intracellular walls of the ethmoids have undergone an atrophic change. If the pathology within the antrum is pronounced and a better conception is desired, the sinus may be filled with lipiodol, and the size of the membrane outlined or the presence of polyps proven. Proetz has given us an easy method of filling the sphenoid and posterior ethmoid cells with lipiodol. The antrum may be filled thru a straight antral needle.

(Fig. 2.) In order to obtain an average standard, I selected one hundred roentgenograms of healthy individuals free from sinus disease and representing the three periods of life. The

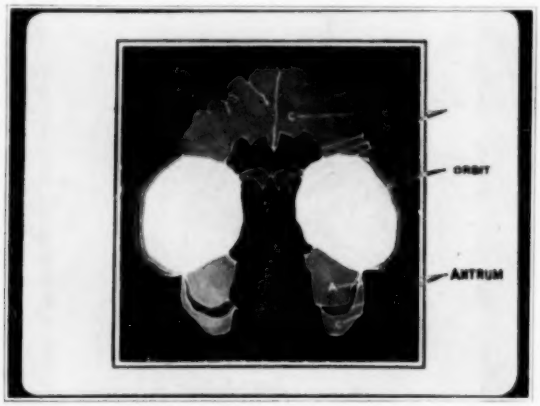

Fig. 2. A. Infantile type; B. Puberty; C. Possible adult limitations.

roentgenograms were taken with the Law technic of 23 degree angle, as this position renders the best anatomic picture. The sinuses of children under three years of age as shown in these roentgenograms are represented by the letter A in Fig. 2. The ethmoids extend up to the level of the cribriform plate: no frontal sinus shadow is present. The floor of the antrum is at a higher level than the floor of the nose. The average shadow of the cases at puberty are represented by the letter B and have the following characteristics: (a) the frontal shadow is uniformly developed and its diameters are relatively equal; (b) the ethmoidal shadow extends upward above the cribriform plate; (c) the antral floor is now on a level with that of the nose. The shadows in the adult roentgenograms may have a wide range of size and still be normal. The breadth of the frontal shadow should exceed its height, and

Fig. 3. Rubber tube into antrum for ventilation.

the floor of the antrum is at a lower level than that of the nose. The shadow of the orbit measures relatively larger at birth than in the adult—in fact, its actual enlargement is immaterial from birth to maturity.

Treatment.

Ventilation: (Fig. 3.) The sinuses are air bearing cavities and are cured by ventilation and not by lavage. This is the reason why dental treatment of antral disease is a failure, for the dentist must depend upon lavage and cannot keep his opening into the antrum from being polluted by saliva and the contents of the mouth. Lavage is of service during the acute stage to rid the sinus of its byproducts of immunization which are toxic, but should be used as sparingly as possible. It is of little value in the treatment of the chronic infections. We are now convinced that the attainment of ventilation is the great value of allowing the rubber tubes to remain in the antronasal wall during the postoperative week.

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DISCUSSION.

Dr. R. C. Lynch, New Orleans, La.: The paper of Dr. Shea opens a large field for discussion. When we think about inheritance in relation to sinus disease, our imagination runs in a good many directions, and the consideration of the absence of a frontal sinus, from a pathologic standpoint rather than an anatomic, is one of the situations which will lead us in all these directions.

From my experience in the epidemics that have visited us, to say that a certain epidemic gives evidence of selection in its action on the sinuses, would be open to question. While bacteria of certain types may have a selective affinity for tissue, and while it is apparently true, and while we do feel that the grippal infection of 1925 affected the bronchial tree more than the sinuses, and that in 1926 affected the ears more than the sinuses or the bronchial tree, and the one in 1927 affected the sinuses —whether this is a scientific fact or whether it is the force of circumstances, needs to be thoroly investigated before we can make such a statement.

I have long been under the impression that tonsillectomy, when done at certain times on certain individuals, has robbed them of something they should have for their protection. At the same time, I would not like to feel that tonsillectomy should not be done in young children because of the possibility of increased sinus infection. In the selected cases that I can pick up in my experience, those patients who were robbed of that something they should have did not necessarily manifest a sinus lesion alone, but manifested other evidences of a loss of that which

might have given them the immunity which belonged to them, if the operation had not been done. On the other hand, Dr. Dean in various papers, concludes that removal of adenoids and tonsils is all that is necessary to clear up the lesions. Now whether he has gone far enough to say that these sinuses remained well after that treatment, or whether the removal under these circumstances decreases their likelihood to reinfection, is another question that should be looked into. Sure it is, that when the sinus is infected, it must be the point of least resistance for reinfection, unless an immunity has been established beyond that which one ordinarily has.

I would like to ask Dr. Shea about the presence of agglutination of secretion as evidence that immunity has been established. That is a new point he brought out, and if he would elaborate it a little, I think it would be of considerable interest. I think it is a rather common experience, that when agglutination of the secretion does occur, the sinus has reached the point of clinical cure, but whether this means that the patient has developed an immunity to the whole situation of that infection, is another question.

The infantile type of sinus is indeed of interest when considered in the light brought out by Dr. Shea, and I believe when we look at our sinuses from that viewpoint, that the small sinus represents previous pathology and diminished resistance, the likelihood of other attacks, or the continuation of the original attack which had never been brought to its proper convalescence, we will be making an advance.

Surely the X-ray study of the sinuses, as outlined by Dr. Shea, is in the right direction, and should be a tremendous suggestion to those men who depend upon the roentgenologist alone for the clinical interpretation of the lesion. I do not believe that the X-ray yet has gotten to the point where it gives us all that we are going to get, but I think there is much more to be done in that direction towards helping us to clear up the unusual case.

Likewise lipiodol has its uses. Surely it can be injected into a sinus and X-rayed to locate pathology; at the same time it can be injected into a sinus which clinically, upon opening the sinus, is absolutely void of any pathology. This has been my experience four or five times, and on a visit to Dr. Mosher's clinic in January, he exhibited some eight or ten plates in which his experience was of the same type.

And lastly, if ventilation, by whatever means obtained, would bring about a cure in all the cases of sinus infection, then our programs for these meetings certainly would be just about one-third as long. In other words, we cannot feel, altho ventilation is of course absolutely essential—we cannot feel that ventilation alone will clear up the pathology entirely.

Dr. R. H. T. Mann, Texarkana, Tex.: I would like to ask Dr. Shea just one question. Is it not possible, that in the children upon whom tonsil and adenoid operations have been performed, sinusitis existed at this time and presented an acute exacerbation only at a later time, when the child took cold? We know that in adults, sinusitis exists oftentimes for five or ten years, or longer, and may never be discovered at all. We also know how easy it is to get lipiodol into the sinuses. Might it not be a fact that these children were infected at the time

of the operation, but resisted the infection until a year or two later, when they took cold and it then developed?

Dr. R. H. Fraser, Battle Creek, Mich.: The deception from the injection of lipiodol lies in an apparent filling defect, which is not justified by pathologic tissue. The most important principle in the avoidance of such deception, is to fill the air spaces in the sinus *after* the removal of all possible discharge by irrigation; and the second principle is to keep the ostium uppermost during the whole of the procedures. This, with regard to the maxiliary sinus, is not difficult. Our patients are sent across the hall to the X-ray with a lateral bend at the waist line, and as much lateral bend of the head as they can manage. Occasionally an individual with arthritis or other disability must be filled on the table in the X-ray laboratory. Deception due to a mass of discharge simulating polypus is avoided by making the 3 mm. opening under the inferior turbinate with the Thorwald antrum drill. Thru this, suction by cannula is applied, and we have been able to withdraw the tenaceous discharge which had resisted the irrigation with saline. These cases may then be refilled, so that the only remaining filling defect actually signifies swelling of the mucoperiosteum. Such precautions have made deception negligible in a series of two hundred observations. Naturally, the findings by this method are not to stand in the shape of diagnoses, independently of history, nasopharyngoscope observations, etc., or regardless of how the technic became modified in its adaptation to the peculiarities of the individual case. By using lipiodol, however, I think we shall not make radical operation of the sinuses unnecessarily, and yet be radical enough to have a high likelihood of direct success. The extent of a hyperplastic sinusitis is difficult to diagnose—even by combining all methods—because, without or even with retained discharge, it may give negative washings. In many cases, opaque injection with lipiodol is essential to diagnosis. Let us recall the disagreeable experience of doing a window operation, which, altho giving perfect aeration and drainage, does not succeed in the cure of second degree thickenings with beginning polypi.

Dr. M. F. Arbuckle, St. Louis, Mo.: Dr. Shea's paper brings up for discussion many important points.

The possibility of an inherited tendency to sinus disease is one which I have long suspected, but regarding which I am still uncertain.

I have families, and I am sure we all have, in which both parents and all of several children are sufferers from chronic sinus suppuration.

I cannot see why or how the removal of diseased tonsils should have a bad effect on the child's resistance to infection. I think with Dr. Shea, that there should always be a definite indication for tonsillectomy before the operation is done. It is my feeling, that many tonsils have been removed in the past without actual indication for the operation.

When adenoid vegetation is of sufficient size to block the nose, it interferes with ventilation of the sinuses and should be removed.

I was much interested in Dr. Shea's report of a case of vertigo due to osteoma of the frontal sinus.

A few years ago, I had a patient suffering with sphenoid suppuration and severe headache, who was also troubled with dizziness. The dizziness was so severe at times, that she would hold on to prevent falling from her chair. When dizziness was not present, it could be

produced at will by rubbing the interior of the right sphenoid with a cotton applicator or a probe. Dr. H. W. Lyman saw the patient in consultation and reported normal vestibular apparatus. This was in November, 1924. She apparently has fully recovered from all her symptoms.

In cases with "persistent infantile" sinuses, it is interesting to note how closely the history of sinus disease coincides with the X-ray findings with regard to the age and incidence of disease.

I think I have been able to confirm Dr. Shea's observations of resumption of growth of a given sinus after infection had been cleared up.

Dr. Joseph D. Heitger, Louisville, Ky.: As Dr. Arbuckle said, this paper brings up a great many points of discussion.

I would like to ask Dr. Shea in regard to this hereditary tendency. Of course we know work has been done on this and it might be said, that as it is a family affair, it is because they eat the same food. Those who want to take up the facial contour proposition might answer it another way, and others who have different ideas might answer it in still a different way.

In regard to the vertigo, I would like to ask Dr. Shea whether this was a real, true turning nystagmus, during the duration of the vertigo, or whether it was an uneasy sensation.

Dr. John J. Shea, Memphis, Tenn. (closing): In answer to the same questions of Drs. Lynch and Arbuckle, concerning the removal of the tonsils and adenoid, I would say to remove the adenoid, but to retain the tonsils to the eighth or tenth year if possible. Maybe one or two attacks of tonsillitis are necessary to maintain an immunity to the infections of the child's environment. Certainly the first attack of tonsillitis is not a sufficient reason for their removal.

If an ordinary head cold is observed, the stage before healing will be marked by a thickening of the nasal discharge. The same may be observed of the antral secretion.

Dr. Mann asks as to the condition of the sinuses of these children at the time of their operation. In as much as I removed the tonsils in the majority of these children, and at that time did not find any symptoms suggestive of sinus disease, the sinuses were apparently healthy.

I agree with Dr. Fraser that the taking of two roentgenograms, one posterior-anterior and the other lateral, will eliminate any mistake caused by air bubbles displacing the lipiodol.

In answer to Dr. Heitiger's question as to the type of reaction, I think that it was a vertigo, but could not produce a nystagmus.

TRANSACTIONS

OF THE

THIRTY-SECOND ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

PRESENTATION OF INSTRUMENTS AND APPLIANCES

A RADIOGRAPHIC ANGLE HEAD TABLE AND RADIOGRAPHSCOPE FOR STEREO SHADOWGRAPHS OF PARANASAL SINUSES, MASTOIDS, ETC.

W. P. Reaves, M.D.

GREENSBORO, NORTH CAROLINA.

X-ray shadowoscopy is the peculiar photographic and optical properties of the X-rays that pass thru the opaque body, and the resistance to the X-rays of its component parts are registered on a sensitized film (shadowgraph).

The greater densities in the same plane prevent the lesser densities from registering; therefore, in making shadowgraphs of the head, mastoids and paranasal sinuses, the head and the cassette must be positioned and angled so that the sinuses and lesser densities are registered. Hence the selective positions of target, patient and cassette in the stereo radiographic unit are obvious.

These positions determine the photographic angles of the X-rays thru the opaque body and thru the sensitized films.

A photograph is a reflected image registered on a sensitized film and printed on a sensitized paper. A shadowgraph is the projected resistance to the X-rays of the component parts of an opaque body registered on a sensitized film by the X-rays which have passed thru the opaque body and film; hence the difference of angulation in reflection and projection in photographs and shadowgraphs to reproduce in stereo a transparent image.

X-ray stereo shadowoscopy is to superimpose shadowgraphs. The marker is in the upper right hand corner, reading position. The shadows of the opaque body displaced to the left is the right shift and is placed in the left box of the stereoscope (2A); the shadows of the opaque body displaced to the right is the left shift and is placed in the right box of the stereoscope (2A). (These positions are reversed because the shadowgraphs are reversed when reflected from the mirrors to the observer's eyes). The shadows from the shadowgraphs in the illuminating-boxes reflected to the ob-

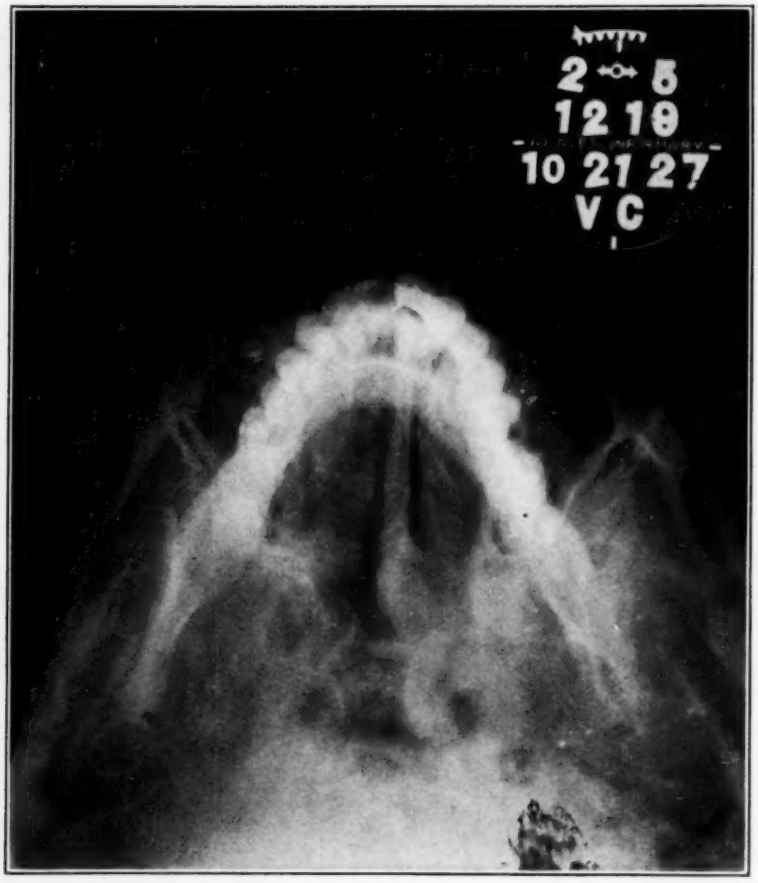


Fig. 1. Stereo X-ray, left shift (showing right purulent sphenoiditis).

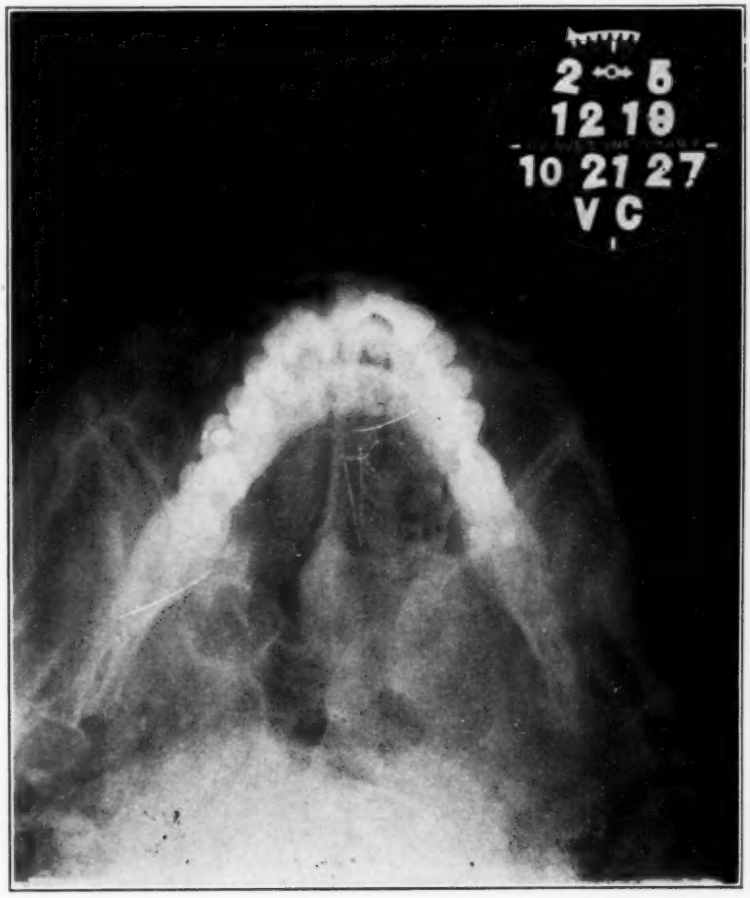


Fig. 1A. Stereo X-ray, right shift.

server's eyes by coordination of illuminating boxes, mirror box and observer's eyes are projected by his optical complex which builds in relief from shadows a transparent image of the opaque body (4A) giving detail and third dimension. Thus it is obvious that to blend the arts of X-ray, photography and optics it is necessary to have a calibrated stereo radiographic unit to fix these positions and angles of target (1), patient (4) and cassette (3) and a marker (Figure 3) to register these selected positions, angles and other data and a calibrated shadowgraphscope (Figure 4) to reduplicate these

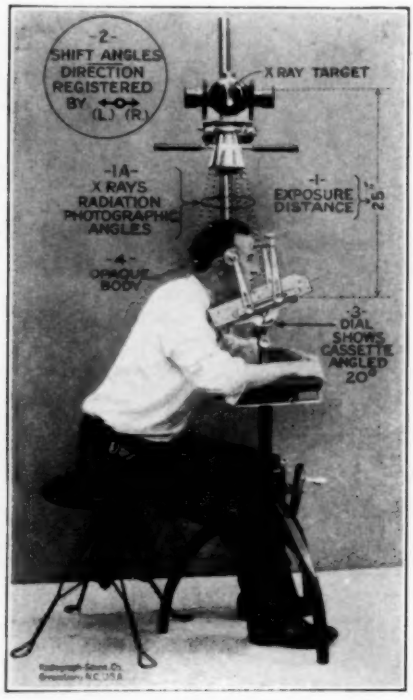


Fig. 2. Calibrated stereographic unit giving positions and angles of patient, target and cassette.

positions and angles to the observer's eyes to build in relief a transparent image of the opaque body. 2A has duplicated shadowgraph positions in reverse to the optical center of the mirror box that cassettes were to target at exposure. 1a has duplicated exposure distance. 3A has duplicated cassette angle. When the observer's eyes duplicate to the shadowgraphs target exposure positions of cassettes, his optical complex builds the transparent image 4A of the opaque body giving true topography, detail and third dimension.

1. Exposure distance.
2. Tube shift.
3. Cassette angle.
Serial Number.
Personal Identification.
Date.
Position

Fig. 3. Marker registering selected positions, angles and other data in Fig. 2.

Fig. 3A. Cover with angle scale to register angle of cassette. (Each point represents 5 degrees.)

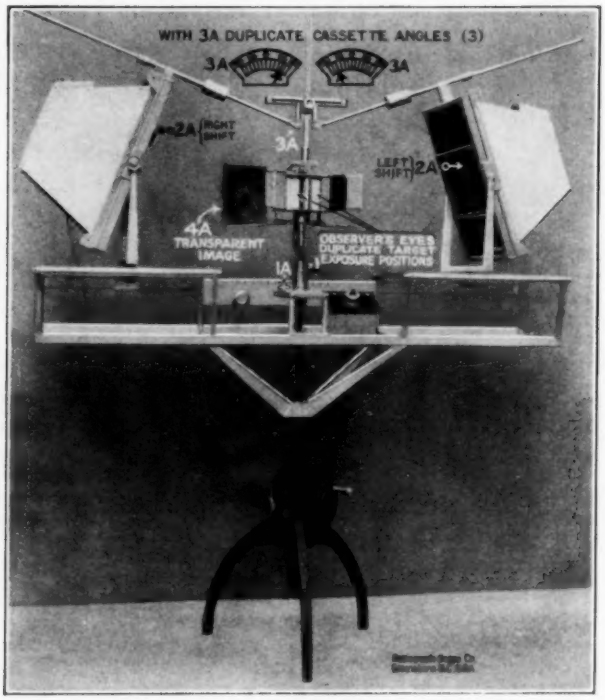


Fig. 4. Calibrated shadowgraphscope to reduplicate the positions and angles of shadowgraphs to the observer's eyes that target was to cassette at exposure.

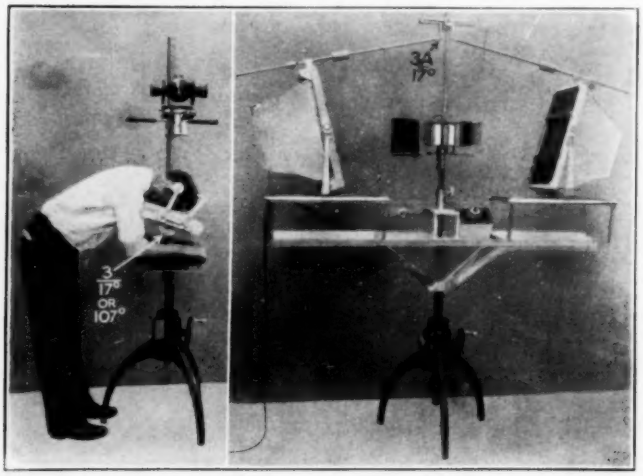


Fig. 5. Cassette angle 3 duplicated in stereo 3A.

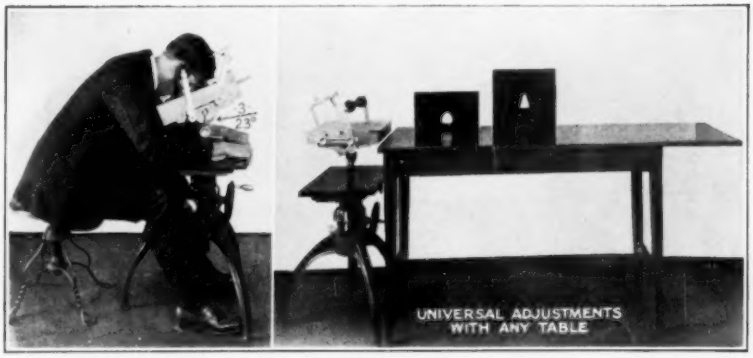


Fig. 6. Shows adaptability of table for any position of patient either sitting or prone.

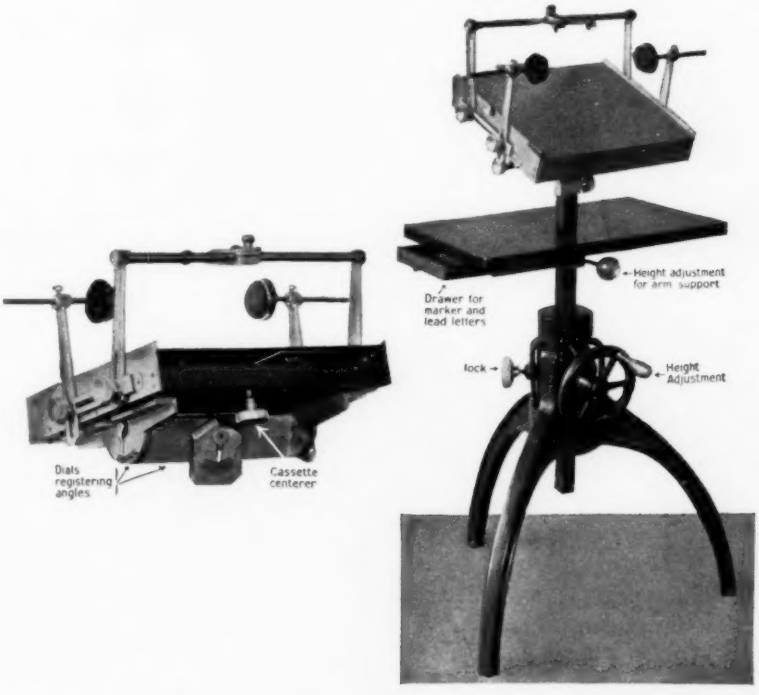


Fig. 7. Stereoradiographic angle head table showing universal adjustment of cassette holder, with dials to show angles, height adjustment to suit the size and posture of each individual patient, arm and head supports to immobilize the patient, eliminating even the vibration of breathing, and lock to give the solidity of one piece.

NEW TEST CARD.

SYLVESTER J. BEACH, M.D.

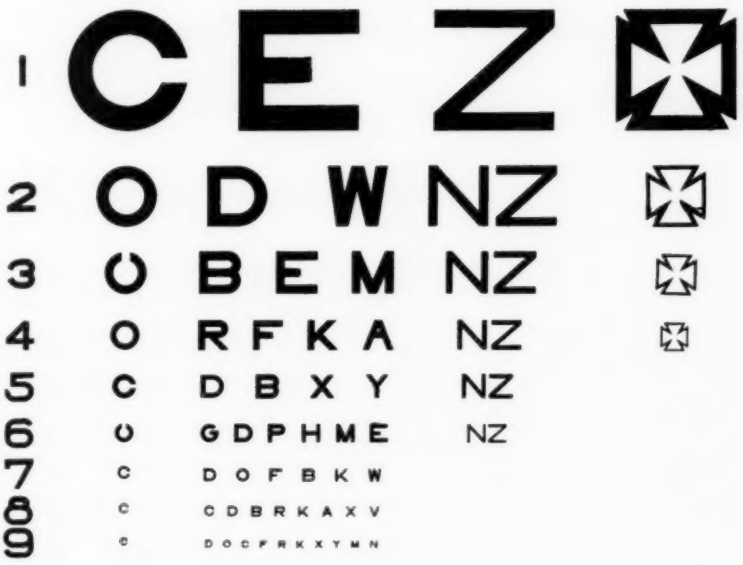
PORTLAND, MAINE.

This test card is based upon the principal I have previously reported[1] namely, that in the letters we use for refracting, the part most sensitive to direction from incorrect lenses is the acute angle. Especially is this the case in examining for the axis and amount of astigmatism. The most useful angular figure is the Maltese Cross. A column of four of these appear at the right edge of this card. They are symmetric, and have a regular interval of 30° between the members.

While in most cases this design gives the quickest and most accurate tests, there are persons who require letters. For them, the combination "NZ" makes a delicate test. "Z" is "N" on its side, so the verticals and horizontals balance each other exactly, as do the diagonals. To the left of the column of "NZs" is a confusion card for checking results. Unlike the cards in common use, composed chiefly of circular letters, the ones here are selected so that at the right edge will be found those letters having acute angles. You will note that "M" and "W" have been restored, greatly increasing the value of the card. Next, to the left, is a column of broken rings, not for refraction but to test the acuity. As my patients like to name these rings I introduced only those that resemble letters, "C," "O," and "U." The numbers are the size of the 6/12 line.

At my hands, this arrangement with a higher proportion of angular and symmetric designs is less fatiguing, and a time saver both for the patient and the examiner.

These cards may be obtained from E. B. Meyrowitz Co., New York City.



REFERENCES.

1. Trans. Oph. Sect. A. M. A., 1927.

INSTRUMENT FOR ENLARGING THE NASAL OPENING.

D. W. Hogue, M.D.

SPRINGFIELD, OHIO.

I wish to present the instrument for the enlarging of the nasal opening in an operation for draining the lacrimal sac into the nose, which I mentioned in the discussion of Dr. Dean's paper. It is a reverse trephine, cutting upwards. The probe is passed down into the nasal opening, and this cuts upwards. It has helped me out of several difficult situations, where the nasal opening was closed. The whole instrument is shown enlarged one-fourth, while the head is shown magnified 6 times to show the cutting edge.

EYE SHIELD FOR MONOCULAR ISOLATION.*

JAMES M. PATTON, M.D.

OMAHA, NEBRASKA.

Presented a shield to be used in cases of ophthalmia—a celluloid shield made to fit the face and adapted to the eye—consisting of a piece of celluloid, X-ray film, 3 in. x 4½ in. It gives protection to the infected eye, and can be cut any size from the largest man to the smallest infant.

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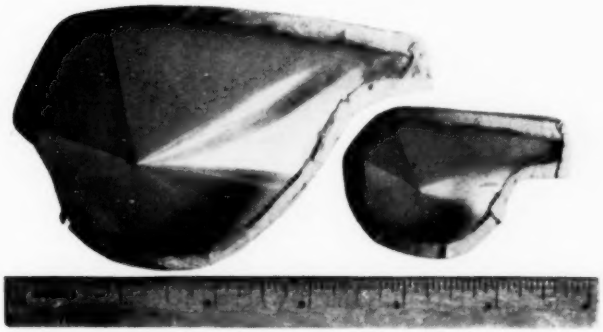
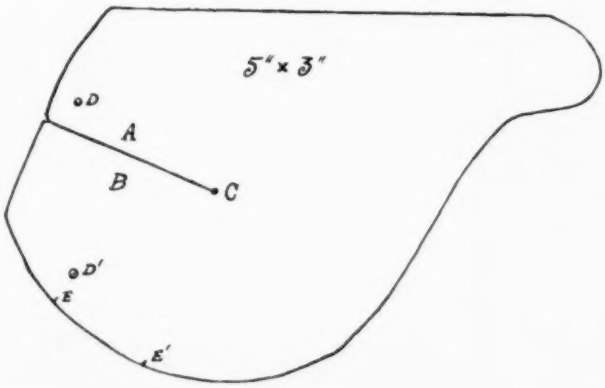
*Thanks are due to the Journal of the A. M. A. for the loan of the cuts.




5" × 3"
D
A
B
C
D'
E
E'

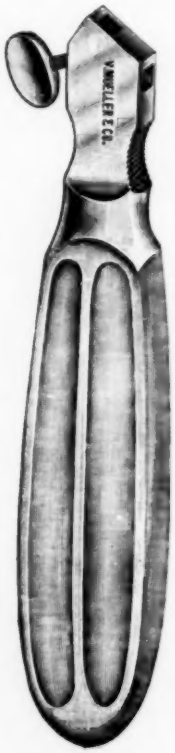
INSTRUMENT FOR BREAKING DOWN ANTRAL WALL.

GEORGE E. HOURN, M.D.

ST. LOUIS, MISSOURI.

This is a very simple instrument for breaking down a resistant nasoantral wall in an ordinary window operation. I think it is valuable only in cases where the wall is quite thick, and where it is difficult to use a punch. You will note it is small and can be readily used where the space is limited.

The idea is to make a perforation with a Wellaminski antrum perforator of any other suitable instrument, and engage the bony partition in the embrasure of the instrument. By a rotary and twisting maneuver of the handle, the bone is readily broken down.



RUBBER CATHETER FOR A BRONCHIAL TUBE.

H. L. Stitt, M.D.

CINCINNATI, OHIO.

I am not exactly presenting a new instrument, but an old proven one in other fields for a new purpose, namely a soft rubber, No. 18, Coude's urinary catheter, as a bronchial tube.

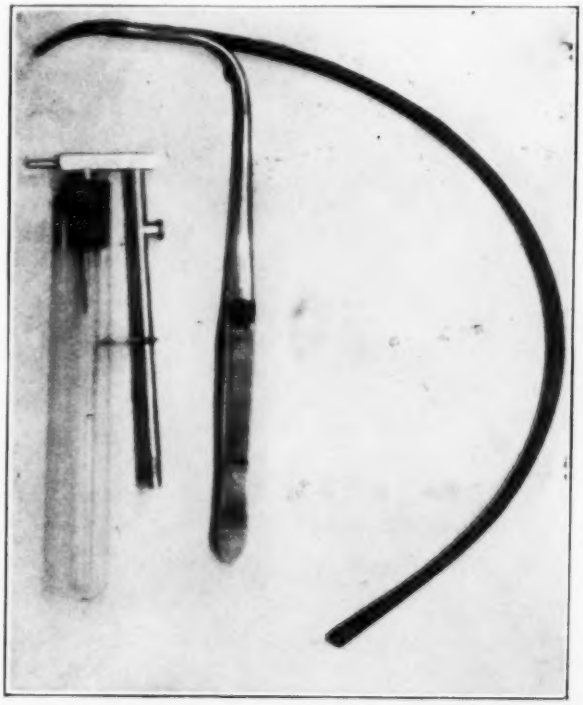


Fig. 1. Coude catheter with Jackson's cotton carrying forceps and Yankauer's specimen collector.

Heretofore, most bronchial aspirations and irrigations have been done with the bronchoscope. Since it is not necessary to place the bronchoscope in immediate contact with the bronchial branches communicating with the abscess or bronchiectatic area, I have conceived the idea, that if a soft rubber

catheter could be directed thru the glottis to the carina by touch, it could be directed into each main bronchus. To accomplish this manipulation, I have employed this No. 18 Coude catheter (patient sitting), introducing it by the indirect method thru the glottis with a Jackson cross action, cotton carrying forceps. The nurse assistant pushes it into the trachea just above the carina; then, by touch, without the aid of an internal semirigid guide or the fluroroscope, it can be directed into either main bronchus. When it is once introduced, an uncontaminated specimen can be obtained with either a Yankhauer or a Clerf specimen collector. Iodized oils

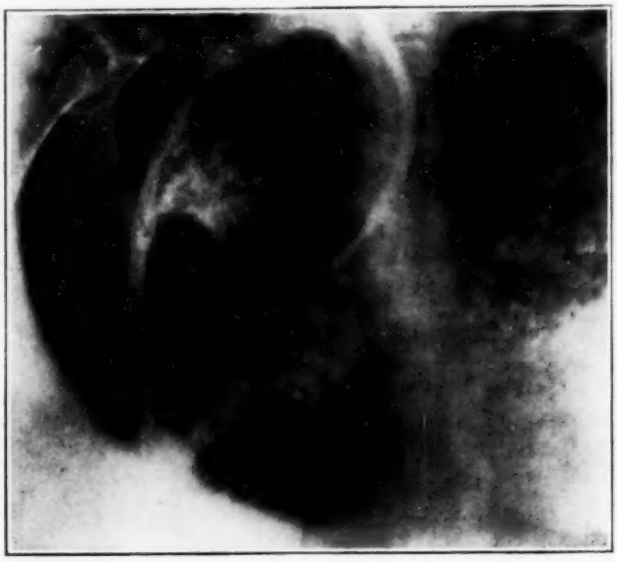
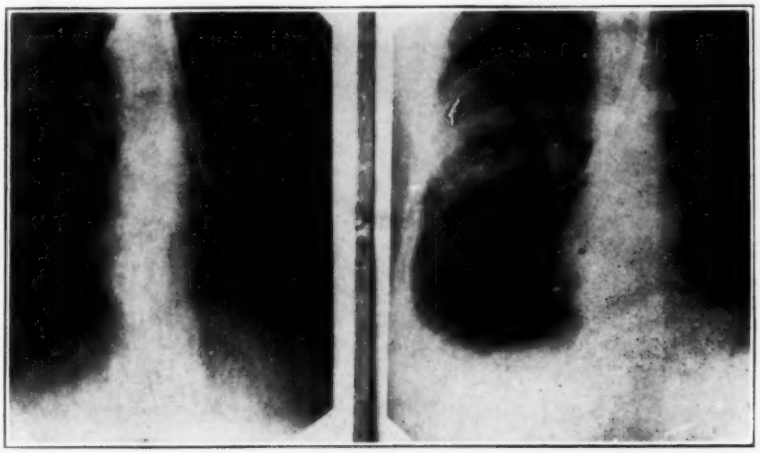


Fig. 2. Catheter placed to lower left lobe abscess.

can be injected into either bronchus at will, or aspiration and irrigation can be performed. I have performed some 1,500 aspirations and irrigations with a hypertonic solution of CAK_2N_a and iodized oil injections by this method without a single mishap, so I feel the procedure is one of merit.

The slides shown here show the catheter in either main bronchus by the method described. I might incidentally say that this case in the beginning was a racemose bronchiectasis, in which I have aspirated and irrigated some twelve to fifteen times, at weekly intervals. The next slide shows the bronchiectasis to be of a cylindrical type. She is free from putrid

expectoration and cough, and apparently clinically well, but has to be lavaged once a month to continue so. My internist and I decided to do a pneumothorax and keep the lung collapsed for a period of six months, to see if this will materially further narrow the diameter of her diseased bronchi. I am sure it will, because we have other cases to warrant this trial.



A B

Fig 3A. Catheter in right main bronchus.
Fig. 3B. Catheter in left main bronchus

TRANSACTIONS

OF THE

THIRTY-SECOND ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

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DEATHS

Dr. Ira Jesse Dunn, Erie, Pa.

Dr. David Isaacs, Omaha, Neb.

Dr. Alfred G. Kruetzer, Milwaukee, Wis.

Dr. Francis Adonijah Lane, Chicago, Ill.

Dr. Frederick Richard Lintleman, Janesville, Wis.

Dr. Hanau Wolf Loeb, St. Louis, Mo.

Dr. Ancil Martin, Phoenix, Ariz.

Dr. William R. Murray, Minneapolis, Minn.

Dr. Joseph M. Patterson, Kansas City, Mo.

Dr. Robert Grigg Reese, New York City, N. Y.

Dr. Charles Moore Robertson, Chicago, Ill.

Dr. Isaac Sernoffsky, Buffalo, N. Y.

Dr. Timothy Joseph Shanahan, Boston, Mass.

Dr. William Sweeny Stucky, Lexington, Ky.

Dr. William Merrick Sweet, Philadelphia, Pa.

Dr. David Emmett Welsh, Grand Rapids, Mich.

MINUTES.

September 13, 1927

The Thirty-Second Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology was held at the Book-Cadillac Hotel, Detroit, Michigan, September 12th to 16th, 1927.

The opening session was called to order at nine-thirty a. m. by the President, Dr. Ross H. Skillern. Following brief words of welcome by Dr. Walter R. Parker, Chairman of the local committee, the President introduced the guest of honor, Gen. Herbert S. Birkett, M.D., Montreal, Canada.

THE PRESIDENT: I now have a most pleasant task to perform, that of presenting our guest of honor. I say present, because it is not necessary to introduce him. Hitherto this Academy has gone out of the country, overseas, for our guest of honor, when all the time we had a man fully deserving of every honor we could give him living right with us. This man, while not flying our flag, is nevertheless an American. He was one of the first to enter the Great War, being chosen as Surgeon General of the Canadian Army. He was one of the first to go abroad, occupying an important position in England and France, and came very nearly making the supreme sacrifice—so near that it was only a question of days—but he rallied, and we are thankful he is with us today.

I therefore present to you our honored guest, surgeon, soldier and scientist, General Herbert A. Birkett, M. D., of Montreal.

BUSINESS SESSION.

The Business Session of the Academy was held on board the steamer "Wauketa" at eight forty-five p. m., Tuesday, September 13th, the President, Dr. Ross H. Skillern, presiding.

Announcements: By Dr. Walter R. Parker, Chairman of local committee.

Reading of Minutes: On motion, duly seconded, the reading of the minutes was dispensed with.

EXECUTIVE SECRETARY'S REPORT.

A great deal of work has been done in the Secretary's office during the past year, brought about in a large measure thru the amalgamation of that formerly done by the Secretary and the Treasurer. Naturally, many controversies arose, and the Secretary wishes to express his personal appreciation to the retiring officers, to Dr. Large and to those members having grievances, for the sincere cooperation exhibited in straightening out their difficulties.

A reasonably accurate system has now been devised to facilitate the routine operation of the Academy, to the end that during the coming year we anticipate but few difficulties. And since the entire official family is entering into the work of the reorganization with a sincere ambition toward cooperation, I feel safe in assuring the membership of continued growth and progress toward scientific advancement.

I want to express my personal appreciation to my associate secretaries, Dr. Benedict, Dr. Myers and Dr. Gradle, to whom entire credit is due for the splendid merit shown in the assembled program to be put on during this meeting.

On July 31, 1927, the end of the firscal year, there were:

Members in good standing	1,368
Honorary Members	12
Life Members	7
One Year Delinquent Members	11
Two year Delinquent Members	5
Total Membership	1,403

During the year there were:

Deaths	17
Resignations	17
Dropped from the roll	7
Total	41

One-hundred and twenty applications for membership were carried during the year, many of whom will be elected to membership at this, the Detroit Meeting.

RESIGNATIONS—1927.

1. Beaumont, John F., Portland, Ore.
2. Bell, Geo. H., New York City, N. Y.
3. Carroll, Frank, Cedar Rapids, Iowa.
4. Cochran, R. W., Madison, Ind.
5. Dworetzky, Julius, Liberty, N. Y.
6. Forbes, H. H., New Yory City.
7. Edwards, Wm. A., Maywood, Ill.
8. Langworthy, H. G., Dubuque, Iowa.
9. Leonard, A. C., Kansas City, Mo.
10. Libby, Geo. F., Denver, Colo.
11. McCall, J. W., Cleveland, Ohio.
12. Means, C. S., Long Beach, Calif.
13. O'Dell, T. T., Aurora, Mo.
14. Starr, Elmer, Mayville, N. Y.
15. Stein, J. C., Chicago, Ill.
16. Stricker, Louis, Cincinnati, Ohio.
17. Wood, Hilliard, Nashville, Tenn.

Respectfully submitted,

W. P. WHERRY,
Executive Secretary.

On motion, duly seconded, the Secretary's Report was accepted.

REPORT OF TREASURER.

PRICE, WATERHOUSE & CO.
Union Trust Building
Cleveland, Ohio.

September 6, 1927.

The American Academy of Ophthalmology and Oto-Laryngology, Cleveland, Ohio.

Dear Sirs:

In accordance with instructions received, we have examined the accounts of your Academy for the period from August 1, 1926 to July 31, 1927, and submit herewith a summary of receipts and disbursements for the period, together with a statement of the assets at July 31, 1927. We also attach a schedule of bonds owned at the latter date.

All receipts and disbursements were traced to the Academy's bank account. The balance in the bank at July 31, 1927 and the subsequent deposit of cash receipts on hand

at that date, were verified by certificate from the bankers, and the bonds on hand July 31, 1927 were counted by us.

Yours very truly,

Price, Waterhouse & Co.

THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNOLOGY.

SUMMARY OF RECEIPTS AND DISBURSEMENTS FROM AUGUST 1, 1926 TO JULY 31, 1927.

Cash in bank, August 1, 1926		$ 8,685.94
Receipts:		
On Income account:		
Dues from members and applications for membership	$15,160.00	
Income from exhibitors	3,059.50	
Interest on bonds, bank balance, etc.	3,755.26	
Profit on bond sold	49.00	
	$22,023.76	
On Capital Account:		
Bond sold—cost	3,951.00	
Total Receipts		25,974.76
		$34,660.70
Disbursements:		
On Expense Account:		
Office expense	$ 4,932.43	
Expense of meetings	7,149.42	
Exhibition expense	1,618.82	
Research work	3,050.00	
	$16,750.67	
On Capital Account:		
Bonds purchased	13,890.00	
Total Disbursements		30,640.67
Cash in bank and on hand July 31, 1927		$ 4,020.03

SUMMARY OF ASSETS JULY 31, 1927.

Cash in bank and on hand	$ 4,020.03
Bonds owned, per schedule	73,971.50
	$77,991.53

THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SCHEDULE OF BONDS OWNED JULY 31, 1927.

Description	Interest	Date Due	Par Value	Cost
Youngstown Sheet and Tube Company Debenture Bonds	6 %	—1943	$ 5,000.00	$ 5,135.00
Union Square Company-Cleveland First Mortgage Leasehold	6½%	—1936	5,000.00	5,000.00
Kansas City Fort Scott & Memphis Railway Refunding Mortgage	4 %	—1936	5,000.00	4,352.50
Chicago, Rock Island & Pacific Railway First & Refunding Gold Bonds	4 %	—1934	6,000.00	5,337.00
Midvale Steel & Ordnance Company Convertible Sinking Fund	5 %	—1936	10,000.00	9,735.00
Swift & Company Sinking Fund Gold Notes	5 %	—1932	5,000.00	4,845.00
American Telephone & Telegraph Company Sinking Fund Gold Debentures	5½%	—1943	4,000.00	4,026.00
Denver Gas & Electric Company General Mortgage	5 %	—1949	4,000.00	3,951.00
Witherbee Shuman Company First Mortgage Sinking Fund—Series "A"	6 %	—1944	5,000.00	4,925.00
Van Sweringen Company First Mortgage & Collateral Trust Notes	7 %	—1930	9,000.00	8,910.00
Southern California Edison Company General & Refunding Mortgage	5½%	—1944	4,000.00	3,865.00
Commonwealth Edison Company First Mort-				

gage & Collateral Series "C"	4½%—1956	5,000.00	4,825.00
Detroit City Gas Company First Mortgage Gold Bonds — Series "B"	5 %—1950	5,000.00	5,025.00
Kingdom of Denmark...	5½%—1955	4,000.00	4,040.00
		$76,000.00	$73,971.50

REPORT OF EDITOR OF TRANSACTIONS.

Sixteen hundred copies of the 1926 Transactions were published at a total cost of $4,161.93. From this should be deducted postage, $345.43; reprints of Directory, $94.00; reprints of Mr. Graves' paper, $35.50; authors' share of cost of cuts, $354.50, leaving a net of $3,332.50, or $2.08 per copy. Of these 1600 copies, 126 were sent to various medical libraries; 120 copies are in the custody of the Editor and printer, and the remainder were sent to the members entitled to them by payment of 1926 dues before March 1, 1926. Back numbers of the Transactions in the custody of the printer are: 1912, 10; 1915, 14; 1917-1918, 6; 1919, 79; 1920, 85; 1921, 103; 1923, 95; 1924, 652; 1925, 175.

The Editor wishes to call attention to the fact that the Transactions were mailed to the members by the middle of April, altho the meeting was as late as September. This was accomplished only as the result of the prompt return of the corrected proof by the respective authors. However, the delay of a few, with the necessity for a thoro revision of the Directory, caused a delay of a month. It is believed that with a little better cooperation, it would be possible to have the Transactions in the mail by March first.

Respectfully,

CLARENCE LOEB,
EDITOR.

On motion, duly seconded, the above report was accepted.

REPORT OF SENIOR MEMBER OF COUNCIL.

The meetings of the Council this year have been characterized by a very marked spirit of cooperation and readiness on the part of all to sacrifice personal and sectional interests to the interests of the Academy.

Thru Dr. Suker we have made arrangements with the Crerar Library of Chicago by which any member of the Academy can have any periodical or book sent to him by paying the transportation charges. He can have bibliographies compiled, abstracts or translations made, in some cases free, in others for a moderate charge. If he wishes to go to the Library to study, he can have special access to the shelves on presentation of his card of membership in the Academy. He can have photographic reprints of articles sent. In short, there is exceptional opportunity for research and study in this, the fourth largest, library in the United States in medical subjects. The arrangement is pursuant to the policy of the Crerar Library to make its facilities available to scientific bodies—for instance, the engineers have been granted similar privileges. The Trustees of the Crerar Library have chosen this Academy as representative of the medical profession, and have offered these privileges to us. The cost to the Academy, it is agreed, will not exceed $1,000; probably not $750.00. It seemed to the Council that this would be a particularly wise expenditure of some of the money available for research.

It has happened that candidates having no certificate from either the ophthalmic board or the oto-laryngologic board, wishing to join the Academy have applied to one of these boards and failed. They have then applied to the other board and passed, and so have been accepted for membership in the Academy. It seems to the Council that this would be properly dealt with by not allowing a candidate who has failed to pass one examination to join the Academy thru the other board. He must pass the board to which he first applied before the Academy will consider his application.

Dr. Casey Wood is recommended as a Life Member.

The meeting place next year will be St. Louis.

The following nominations are made:

President	Luther C. Peter	Philadelphia
President-elect	Harris P. Mosher	Boston
1st Vice-President	Frank L. Dennis	Colorado Springs
2nd Vice-President	Lawrence Post	St. Louis
3rd Vice-President	H. H. Sanderson	Detroit
Executive Secretary	W. P. Wherry	Omaha
Treasurer	Secord H. Large	Cleveland
Editor of Transactions	Clarence Loeb	Chicago

Member of Council for 3 years	John M. Wheeler	New York
Member American Board for Ophthalmic Examinations	James M. Patton	Omaha

Associate Secretaries will be appointed by the incoming President.

WALTER B. LANCASTER,
SENIOR MEMBER OF COUNCIL.

The several items of the above report were taken up and voted upon seriatim, and the report adopted.

REPORT OF COMMITTEE ON ARRANGEMENTS.

There is very little report, this being a business session, except that I would like to speak a word of appreciation for the courtesies extended to our Committee by the hotel authorities. We have had all the floor space we wanted given to us without any charge whatever.

I would also like to speak a word of commendation to the Bureau on Conventions of the City of Detroit. They have furnished all the extra clerks in the registry office without any cost to us.

WALTER R. PARKER,
CHAIRMAN.

REPORT OF PROGRAM COMMITTEE.

The Program Committee, consisting of Drs. Skillern, Woodruff, Parker, Benedict, Myers, Gradle, and the writer, met in Chicago, December 18, 1926, and after careful deliberation constructed a nucleus for the scientific program to be given at this meeting. Instructional Conferences as suggested by Dr. Gradle were carefully gone over, and in a large part those Conferences to be given were determined upon.

Another meeting was held in Washington, D. C., during the recent session of the American Medical Association, at which time the program as now presented was completed.

The Program Committee respectfully requests that those wishing to present papers next year kindly submit title and brief abstracts to the Secretaries of the respective sections prior to December first next, since the program for next year

will in part be drafted some time during that month. At any event, subjects should be offered to the committee prior to the Minneapolis meeting of the American Medical Association.

Respectfully submitted,

W. P. Wherry,
Chairman.

Report of Committee on Tuning Forks and Tests of Hearing.

In 1925, your committee submitted a rather detailed report, together with a few additional facts in 1926. During the past year, we have been unable to develop any new ideas along the lines of functional hearing, but we have something to report regarding a new type of fork.

You will recall that one of the essentials of good forks proposed by your committee was the use of a metal that would not rust. When a fork is nickeled, the nickeling sometimes peels and adventitious sounds are produced by the loose pieces of metal. On the other hand, unless a steel fork is very carefully handled and properly oiled, it tends to rust and in time to change pitch. Various metals have been produced at various times, but as a rule these have not proven satisfactory. Thru the courtesy of Colonel Fabyan of the Riverbank Laboratory, your chairman was presented with a fork made of an alloy of magnesium, of which this metal constitutes about 99 per cent. of the mixture. This gives the fork an extremely light weight, which is very easily handled. It is to be remembered that the specific gravity of steel is about 7, that of aluminum 2.7, and that of magnesium only 1.7.

This magnesium fork has a vibration period almost as long as that of a good steel fork, is very easily manipulated, is rust proof, and the cost of production will probably not be greater than that of the steel forks. Magnesium forks are superior to commercial forks in pitches from about 256 to 2000 double vibrations. At the higher rates, the magnesium forks are about as good as the steel ones, but no better. The lowest feasible magnesium forks are about 100 double vibrations. Below that point, they are not of much use, due probably to damping by the air. We have noticed, with the light weight magnesium fork, that there is much less bone conduction than with the steel fork. The reason for this is that most

of the energy goes back into the fork rather than into the head, as it does with the heavier forks.

It is too early to state definitely whether this fork will be of as practical use as steel forks, since we do not know as yet how brittle this alloy is, and therefore how easily the forks may be injured. It will take considerable experimentation to definitely determine these various points, but we believe that a great advance has been made in the direction of obtaining a rustless, light weight fork.

The committee at this time merely reaffirms the recommendations made in the reports of the previous years, which are in the archives of the Academy, and desires to be continued another year, in order to further investigate facts bearing upon the standardization of tuning forks and tuning fork tests.

Respectfully submitted,

L. W. Dean
George W. Mackenzie
E. G. Gill
Robert Sonnenschein, Chairman.

Report of Otolaryngologic Board.

Your Board has held three meetings this year. At present we have certificated a little over one thousand applicants. At the meeting yesterday, we had 102 applicants. We had a very successful meeting, thanks to Dr. Shurly and his staff. We had plenty of patients and were very well taken care of, and I wish the Academy would give Dr. Shurly a vote of thanks for his excellent work.

Dr. Wherry, who is the other member from this society, is now the Secretary of the Board. He was elected to take Dr. Loeb's place.

Respectfully submitted,

T. E. Carmody.

Report of Ophthalmic Board.

During the year, the office of the Secretary of this Board has prepared a directory of those who have been certificated, which can be obtained from the office of the Secretary for $1.25.

Respectfully submitted,

J. M. Patton.

REPORT OF COMMITTEE ON PUBLIC SERVICE AND LEGISLATION.

As to the reason for this committee: A few years ago, there was a national committee formed on which this society was to have representation. The national committee has not, as far as we know, functioned during this year. Your committee consists of Dr. Shea and myself. We have met three times, but we have not heard from the central committee.

We, therefore, submit for your consideration our report, for your committee feels that it can be of considerable help, first, in drawing general attention to the activities of the society by stating the aims of the organization, and especially stressing the size of this society. We feel that one of the great benefits will be the improvement of the service rendered individual members.

Legislation: The society already has several subcommittees on legislative affairs. One has just changed its name today, and the other is the committee on lye legislation.

This is a national committee, and therefore the things that come before us should be of national scope; but before we can reach the headquarters of the nation, we must have an understanding of what is going on in our different states and localities, and therefore we draw your attention to the motor acts of the various states, especially those having to do with ophthalmology. In many states a man, before he can secure his driver's license, must stand a test. In New York it is a visual test, and then some man takes you around the block to see if you can drive your car.

The committee especially commends the action of the editorial boards of the American Journal of Ophthalmology and the Journal of the American Medical Association. Those of you who have been reading appreciate that both of these journals have given this society very excellent preliminary notices, and the very efficient secretary of the Secretary, Miss McGovern, informs me that during the last month many have written in asking how they may become members of this active and powerful society.

In the past the local papers have carried ridiculous accounts of scientific meetings. This year your committee is endeavoring to censor all newspaper accounts, in the hope of allowing only authentic reports to be published.

We feel that in the last year your committee, functioning

for the first time, has found some of the things that should be done.

Respectfully submitted,

ARTHUR J. BEDELL.

REPORT OF COMMITTEE ON OPHTHALMIC AND OTOLARYNGITIC PATHOLOGY.

The Committee on Ophthalmic Pathology, working in conjunction with the Academy, is continuing the growth of its work. We are receiving specimens at the Army Museum in Washington at the rate of about 600 a year, and to date we have more than 2,000 museum specimens representing various conditions in ophthalmic pathology, collected for the most part by members of the Academy without cost. In return for this donation of specimens, we receive a pathologic report as well as a histologic section of the material sent in.

The work has now grown greater than the Museum can handle with the limited facilities afforded them by Congress. The museum is part of the Army Museum under the Army Medical Department, and the appropriation for that department is being cut down by Congress. As a result, it becomes absolutely essential that help from this Society be now forthcoming. Why is this help necessary? Because the museum is just beginning to be in a position to accomplish what they wish to do. One of the great aims of the Museum is not only to be a true pathologic museum, but also to put out study sets—sets of fifty or one hundred slides representing varied conditions of ophthalmic pathology, these sets to be loaned to members of the Academy at no cost beyond transportation, for a period not to exceed three months. Each slide is accompanied by a complete description, and a man desiring to improve his knowledge of pathology can borrow one of these sets from the Museum, and in addition receive any other information the Museum has, on request.

If the aims of the Committee on Ophthalmic Pathology be carried out, fianancial assistance in the amount of about $500 for this year should be given the Museum. The Section on Instruction in the past has been paying its own way by the fees from registrants. Owing to the character of the expense in the past, the expense has been far greater than it is for this year. This year it is expected that the Section on Instruction will show a profit of $1,000, and of that, $500 could well be

given to the Army Medical Museum, keeping the promise made by this society five years ago.

Respectfully submitted,

HARRY S. GRADLE.

REPORT OF AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY.

This change in the committee was completed by the action of the Academy a year ago, and it was also upheld by the Section on Ophthalmology of the American Medical Association in May. At that meeting, certain recommendations were adopted. These recommendations have been accepted by the Section on Ophthalmology of the American Medical Association, by the American Ophthalmological Society, and in effect, this evening, by accepting the report of the Council. The specific recommendations are included in this report.

The recommendation to make this committee permanently representative of the three American organizations of ophthalmologists, is based on an understanding that has developed of the wide scope and importance of the work that might be done thru it to educate the medical profession and the public.

1. As to the need that grows out of the exacting use of our eyes, that become very common under the requirements of modern civilization, for exact optical adjustment to their work.

2. As to the intimate and dominant influence that optical adjustments and other conditions of their use exert on the health of the eyes and the general health of those who have to use them.

3. As to the interdependence of ocular and general health that renders necessary the broad understanding of ocular physiology and health on the part of all who can wisely advise with regard to such health matters, as well as familiarity with the general signs of ocular or related diseases.

The carrying forward of such education is a duty on the part of the members of this society. The extent of the understanding makes it necessary that for years to come details with regard to it, relations of those who claim to be working in the direction of the common good, the developments of science and the perfecting of arts relating to it, must demand thoughtful, conscientious, continued attention to it on the

part of all engaged in the care of the eyes and their representatives. There are important matters, some already under consideration, that must be closely studied and broadly considered, that may well be subjects for future reports from this committee.

The committee requests that the name, (THE AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY) be accepted to designate this committee by the American Academy of Ophthalmology and Otolaryngology.

The committee also requests that it be continued as a permanent national committee—three members to be elected this year, one to serve one year, one two years, and one three years, and that each succeeding year one new appointment shall be made, this appointee to serve for three years.

JAMES M. PATTON
CHARLES A. BAHN
EDWARD JACKSON, CHAIRMAN.

REPORT OF COMMITTEE ON LYE LEGISLATION.

The committee—Dr. Stucky, Dr. Arbuckle, Dr. Hampton and myself—have been informed that since the law was passed the committee is of no value. However, we could not make a final report without knowing just how the matter stood, consequently I wrote to Dr. Chevalier Jackson, and on August 11th received this reply:

"Please call up the printing department of the Journal of the A. M. A. and ask them to send you the galley proof of the report of the Committee on Lye Legislation. The report may not be printed by the date you mention. If you are unable to obtain the galley proof, I shall be very glad to send you a typewritten copy.

"Briefly, we have obtained Federal legislation in 17 states. It is of the utmost importance, however, that legislation be passed in all of the remaining states, and Illinois is one of the most important of these.

"I would strongly urge that you get in touch with Dr. W. C. Woodward of the Bureau of Legal Medicine, A. M. A., Chicago, and inaugurate a campaign for lye legislation in Illinois.

Yours very truly,
Chevalier Jackson."

I wrote to Dr. Woodward, who was out of town at that time, but I received the galley proof, and you will read it in the Journal. Dr. Woodward has answered my request, and we have a meeting October 3rd, so that is the time when this matter will be taken up. While the report is, that the law has been passed, it is evidently not entirely so. We would ask that the committee be continued.

Respectfully,
JOSEPH C. BECK, CHAIRMAN.

ABSTRACT OF REPORT OF NATIONAL COMMITTE ON LYE LEGISLATION.

Your committee begs leave to report that a federal law providing for proper labeling of packages of household lye and other caustics has been enacted.

It is essentially the same as the model law drafted by Dr. W. C. Woodward.

Support for its passage was received from the lye packing interests, who asked for only two very reasonable requirements, one concerning a technicality and the other that this committee endeavor to secure uniformity in the provisions of state legislation.

Your chairman wishes to state that Dr. Woodward and all members of this committee did all that lay in their power to further the enactment of this piece of federal legislation; but it is only fair to state that all our efforts would undoubtedly have been fruitless had it not been for the work of Vice Chairman Dr. Charles W. Richardson.

Thru the efforts of Dr. J. E. Ellegood, with the aid of those of the members of the profession whose support he obtained, Delaware now has a law closely following the model law drafted by Dr. Woodward.

Thru the efforts of Dr. E. M. Seydell and the members of the profession whose support he obtained, a law conforming closely to the model law was enacted in the state of Kansas.

Dr. Francis P. Emerson elicited the cooperation of the profession and legislators in Massachusetts to the extent required to enact a law closely conforming to the model law drafted by Dr. W. C. Woodward. The prestige of Massachusetts law with attorneys and legislators everywhere makes this success an especially important aid to our future work. Dr. Emerson calls attention to the importance of "having had

figures to show the senators that not only were there actual cases occurring, but to give them figures of how many there had been the preceding year."

The activities of the members of this committee up to the present time have resulted in the enactment of laws providing for the use of the word "POISON," conspicuously printed, on all packages of household lye in the following states: Pennsylvania, Florida, New Jersey, South Carolina, Iowa, Vermont, Oregon, Nevada, New Hampshire, West Virginia, Colorado, Minneapolis, Louisiana, Virginia, Delaware, Kansas and Massachusetts.

Good work is now being done by our committee members in every state of the Union in which lye legislation has not been enacted. In some states success is imminent, in other states, peculiar local obstacles resulted in defeat for the time being. Success in these states is only a matter of time. No legislator who has seen the evidence of poisoned children is or could be opposed to the fundamental principle of a warning label. CHEVALIER JACKSON, CHAIRMAN.

REPORT OF THE COMMITTEE ON HARD OF HEARING.

Your committee begs to report progress during the past year in several activities within its field. This progress has been attained thru the cooperation of its individual members with the members of committees of like name and purpose of our other national otolaryngologic societies, and with the members of committees of the American Federation of Organizations for the Hard of Hearing.

Chief among our activities has been a campaign of publicity to acquaint the medical profession and the laity, particularly the educator, with modern methods of preventing deafness, and to interest these groups in the work of testing the acuity of hearing of school children. The purpose is to detect as early as possible in the school child conditions which, if neglected, might lead to severe hearing loss; to give the school child the benefit of timely preventive or corrective treatment, and to afford him the best possible educational opportunities according to his specific needs. This often means only the appreciation on the part of the teacher that a given child requires a front seat in the school room, or should be given instruction in lip reading, or in severe cases, be assigned to a class for special instruction for the hard of hearing, the ob-

ject being to avoid as far as possible retardation in his school work.

One year ago, only two or three cities in the United States had officially adopted the periodic examination of the hearing acuity of its public school children by audiometric methods. Today, between thirty and forty cities have put this system in practical operation and many more are about to do so. During the past year, various educational and scientific societies have passed resolutions endorsing this work, among them being the American Student Health Association, the Minnesota State Medical Society, and the Minnesota Academy of Ophthalmology and Otolaryngology. The attention of other national organizations needs to be called to this work, when they, too, will gladly take similar action.

The Committee of the American Medical Association on the Hard of Hearing Child, of which Dr. Harold Hays is chairman, has begun the expansion of its work by appointing an interested otologist in each state as a member of its subcommittee, to exercise a local leadership in these activities.

The past year has witnessed the successful inauguration of "Hearing Week", to be nationally observed by directing the attention of the public to the need of preventing deafness and conserving the hearing, and to the methods employed in this work as it applies to the rehabilitation of the hard of hearing as regards their educational, economic and social welfare. The public press and our broadcasting stations have generously given their aid in this effort.

A worthwhile contribution, in which members of your committee have had a part, has been the compilation of a comprehensive report by the commission of the American Federation of Organizations for the Hard of Hearing, recently published and given a wide circulation by the Bureau of Education of the Department of the Interior.

A further noteworthy step is represented in the report by Dr. Douglas MacFarlan of the Special Committee of the American Federation, dealing with the work of the Bureau of Standards in obtaining accurate data covering the real capacity and efficiency of various electrical aids to hearing. This report was read before the American Otological Society at its last meeting and affords valuable information for your committee, for the otologist, and for those afflicted with any considerable degree of hearing loss.

In some communities, however, these handicapped people, working eagerly for their fellows, feel the need of the help and counsel of the otologist, who, by virtue of his training and understanding, is preeminently fitted to aid them. Tho he cannot cure their hearing defects, they still regard him as their natural friend, and look to him for advice and encouragement in their endeavors. Public interest in this work for the hard of hearing is growing at a surprisingly rapid rate. The wise otologist will not allow himself to become conspicuous or to be misunderstood by his apparent indifference.

It is the earnest recommendation of your committee that each Fellow of the Academy who practices otology interest himself actively in his own community in some phase of this work for the hard of hearing.

Respectfully submitted,

HOWARD V. DUTROW
HAROLD HAYS
CHARLES W. RICHARDSON
GEORGE L. TOBEY
CLAUDE T. UREN
HORACE NEWHART, CHAIRMAN.

REPORT OF COMMITTEE ON INSTRUCTION.

Your Section on Instruction has very little to report, as the work is already published. You will be allowed to judge for yourselves, beginning tomorrow.

This year the instructional work is of somewhat different character, and we have been led to this by the evident desire for this kind of work expressed in the past, and we trust you will say how you like it—whether you prefer the conference type of presentation, or would rather go back to the didactic type. The committee can work only with the valuable assistance of the members of the Academy, and we can only do so if you will be free in your criticism, both constructive and destructive. Of course we prefer the constructive type.

The work of making up a series of conferences of this type, nearly sixty, is very difficult, both as to material and as to leaders of the conferences, and your committee would appreciate most deeply the help you can give us by suggesting material for conferences, or in suggesting the names of leaders.

Respectfully,

HARRY S. GRADLE, CHAIRMAN.

Moved by Dr. W. B. Lancaster that the foregoing reports of special committees be accepted, and the committees continued. Motion seconded and carried.

Moved by Dr. Allen Greenwood that the Academy donate $500.00 to the Army Medical Museum, in view of the recommendation of the Committee on Pathologic Work. Motion seconded and carried.

Report of Committee on Necrology.

The names of those who have left us are:

Dr. Ira Jesse Dunn.....Erie, Pennsylvania
Dr. David Isaacs.....Omaha, Nebraska
Dr. Alfred G. Kreutzer.....Milwaukee, Wisconsin
Dr. Francis Adonijah Lane.....Chicago, Ill.
Dr. Frederick Richard Lintleman.....Janesville, Wisconsin
Dr. Hanau Wolf Loeb.....St. Louis, Missouri
Dr. Ancil Martin.....Phoenix, Arizona
Dr. William B. Murray.....Minneapolis, Minnesota
Dr. Joseph M. Patterson.....Kansas City, Missouri
Dr. Robert Grigg Reese.....New York City, N. Y.
Dr. Charles Moore Robertson.....Chicago, Ill.
Dr. Isaac Sernoffsky.....Buffalo, New York
Dr. Timothy Joseph Shanahan.....Boston, Massachusetts
Dr. William Sweeny Stucky.....Lexington, Kentucky
Dr. William Merrick Sweet.....Philadelphia, Pa.
Dr. David Emmett Welsh.....Grand Rapids, Michigan

I wish the President would ask you to stand with bowed heads for a moment in memory of those who have passed away.

Respectfully,
T. E. Carmody, Chairman.

Dr. Walter R. Parker: If it is in order I should like at this time to speak of the death of Dr. Eugene Smith, who was one of the earliest members of the Academy, tho not a charter member. His death occurred yesterday and his funeral will be tomorrow at two o'clock. Out of respect to his memory and as an expression of sympathy from this society, I would move that the Secretary be instructed to send flowers to the funeral at two o'clock tomorrow afternoon.

Motion seconded and carried.

Unfinished Business.

The Secretary: The following proposed changes in the By-Laws of the Academy have been suggested:

"Article 3, Section A: Change the wording in the latter part of second paragraph to read:

"If an applicant for fellowship does not present a certificate from either of the examining boards within three years from the date of his application, the application fee is forfeited; however, the applicant may renew his application for an additional three years without paying an additional fee."

"Article 4, first paragraph: Under 'Officers' change the word 'General' Secretary to 'Executive' Secretary."

These changes were voted on separately, and unanimously carried.

Dr. E. C. Ellett: During this meeting one of the oldest members of the Society, who was in attendance, who is a past President of the Academy, Dr. W. L. Dayton of Lincoln, Nebraska, was taken so ill that it was necessary to take him to a hospital. I move that the Executive Secretary be instructed to send a message of sympathy to Dr. Dayton in his illness, and our wishes for his speedy recovey.

Motion seconded and carried.

Dr. Harry S. Gradle: I would like to present a motion for the appointment of a new committee. I would like to move that the incoming President appoint a committee, of the size he may see fit, to investigate the merit of many of these socalled "cures" for incipient cataract.

Motion seconded and carried.

Election of Members.

The Secretary: The following candidates, who have been certificated by the Boards, are recommended for your consideration:

Beach, Dr. Bennett Sheldon, 76 W. 86th St., New York,Op.

Beck, Dr. August Leo, 421 Huguenot St., New Rochelle, N. Y., OALR.

Black, Dr. Paul, 514 First Nat. Bank Bldg., Lincoln, Neb., OALR.

Bouvy, Dr. Leo Benedict, La Grande, Oregon, OALR

Brown, Dr. Chas. Wm., 613 Commonwealth Bldg., San Diego, Cal., ALR.

Burke, Dr. John Robert, 131 Chestnut St., Holyoke, Mass., OALR.

Cohen, Dr. Herman B., "The Lenox", Spruce and 13th Sts., Philadelphia, Pa., ALR.

Davies, Dr. Josiah S., 704 St. Helen's Ave., Tacoma, Wash., OALR.

Davis, Dr. Frederick Allison, 122 W. Washington Ave., Madison, Wis., OALR.

Doherty, Dr. Wm. Brown, 150 W. 55th St., New York, N. Y., OP.

Durr, Dr. Samuel A., 617 Electric Bldg., San Diego, Cal., Op.

Ellis, Dr. Zenas Horace, 30 W. 59th St., New York, N. Y., Op.

Figi, Dr. Frederick Adam, Mayo Clinic, Rochester, Minn., ALR.

Fink, Dr. Walter H., 426 LaSalle Bldg., Minneapolis, Minn., OALR.

Flynn, Dr. Chas. T., 1138 Chapel St., New Haven, Conn., ALR.

Folsom, Dr. Wm. H., 395 E. Division St., Fond du Lac, Wis., OALR.

Freimuth, Dr. Louis Elmer, 234 University Club Bldg., St. Louis, Mo., ALR.

Galloway, Dr. Thomas Cyrus, 636 Church St., Evanston, Ill., ALR.

Grant, Dr. Lee E., 823 David Whitney Bldg., Detroit, Mich., OALR.

Gray, Dr. E. B., Mercer Bldg., New London, Conn.

Haseltine, Dr. Burton, 122 S. Michigan Ave., Chicago, Ill., OALR.

Higbee, Dr. David Rathburn, 409 Watts Bldg., San Diego, Cal., ALR.

Hoffman, Dr. John N., 308 Brant Bldg., Canton, Ohio.

Hoffman, Dr. Louis George, 30 N. Michigan Ave., Chicago, Ill., OALR.

Hughes, Dr. John A., 1220 Exchange Bldg., Memphis, Tenn., OALR.

Jones, Dr. Ulysses Grant, Johnson City, Tenn., ALR.

Lebensohn, Dr. James E., 3159 W. Roosevelt Rd., Chicago, Ill., OALR.

LeCompte, Dr. Edward Dexter, 627 Boston Bldg., Salt Lake City, Utah, OALR.

Lejeune, Dr. F. E., 632 Maison Blanche Bldg., New Orleans, La., ALR.

Levengood, Dr. H. Wilson, 327 Wilshire Bldg., Santa Monica, Cal., OALR.

Leyda, Dr. James Harold, 406 Metropolitan Bldg., Denver, Col., ALR.

Lundon, Dr. Arthur Edward, 119 Crescent St., Montreal, Can., ALR.

Lyle, Dr. Donald Johnson, 19 W. 7th St., Cincinnati, Ohio, OALR.

McDannald, Dr. Clyde E., 100 W. 59th St., New York, N. Y., Op.

McWilliams, Dr. Chas. Allen, Gulfport, Miss., ALR.

Mellinger, Dr. Wm. J., 120 E. Sola St., Santa Barbara, Cal., ALR.

O'Rourke, Dr. Donald Hunter, 217 Imperial Bldg., Denver, Col., Op.

O'Sullivan, Dr. Timothy Joseph, 10 Pine St., Portland, Me., ALR.

Phelps, Dr. Haskell Sidney, 114 W. 13th St., New York, N. Y., ALR.

Poos, Dr. Edgar E., 13300 Woodward Ave., Detroit, Mich., OALR.

Quincy, Dr. Josiah E., 270 Commonwealth Ave., Boston, Mass.

Ryan, Dr. Linus M., Wall Bldg., St. Louis, Mo., ALR.

Savage, Dr. Moses M., 1720 Eutaw Place, Baltimore, Md., OALR.

Schall, Leroy A., 520 Commonwealth Ave., Boston, Mass., ALR.

Schuster, Dr. Frank P., 403 First Nat. Bank Bldg., El Paso, Texas, OALR.

Schwartz, Dr. Alfred A., 203 W. 20th St., New York, N. Y., ALR.

Skirball, Dr. Joseph Jacob, 353 Commonwealth Ave., Boston, Op.

Smith, Dr. Carlie Souter, 214 N. Jefferson Ave., Springfield, Mo., OALR.

Stark, Dr. Wm. Berkeley, Mayo Clinic, Rochester, Minn., ALR.

Stoutenborough, Dr. Wm. A., 4816 Pine St., Philadelphia, Pa., OALR.

Swerdfeger, Dr. Elbert Byron, 1008 Republic Bldg., Denver, Col., ALR.

Van Meter, Dr. Eugene Robt., 817 University Club Bldg., St. Louis, Mo., ALR.

Veasey, Dr. Clarence Archibald, Jr., 404 Paulsen Bldg., Spokane, Wash., OALR.

Wagner, Dr. Wm. Alfred, 1003 Pere Marquette Bldg., New Orleans, La., ALR.

Weinstein, Dr. Morris A., 1309 Spruce St., Philadelphia, Pa., ALR.

Williams, Dr. Mervyn Morgan, 148 Adams St., Scranton, Pa., Op.

Winger, Dr. Ira B., 225 Michigan St., Toledo, Ohio, OALR.

Woodruff, Dr. Walter Stuart, 421 Huguenot St., New Rochelle, New York, ALR.

Not Certified by Boards until after program was printed:

Abrahamson, Dr. Ira A., 605 Traction Bldg., Cincinnati, Ohio, OALR.

Ambrose, Dr. Elmer Cleo, Logan Bldg., St. Joseph, Mo., OALR.

Boone, Dr. Jesse Franklin, 30 N. Michigan Blvd., Chicago, Ill., OALR.

Bushman, Dr. Louis B., 631 City Nat. Bank Bldg., Omaha, Neb., OALR.

Brown, Dr. Audrey Orin, 742 Maccabee Bldg., Detroit, Mich., OALR.

Campbell, Dr. Malcolm Duncan, 1613 David Whitney Bldg., Detroit, Mich., OALR.

Clifford, Dr. Thomas Percival, 1320 David Whitney Bldg., Detroit, Mich., OALR.

Embery, Dr. Frank, 4660 Frankfort Ave., Philadelphia, Pa., ALR.

Ferguson, Dr. Robt. J., 59 College St., New Haven, Conn., OALR.

Fraser, Dr. Wilbert Grieve, 52 Nepean St., Ottawa, Can., OALR.

Greenfield, Dr. Samuel D., 169 New York Ave., Brooklyn, N. Y., ALR.

Gallagher, Dr. John H. C., 317 Main St., Springfield, Mass., ALR.

Hallett, Dr. Geo. De Wayne, 274 W. 86th St., New York, N. Y., Op.

Jones, Dr. Wm. Solomon, 232 Sheridan Road, Menominee, Mich., OALR.

Keating, Dr. Thomas Francis, 5057 Woodward Ave., Detroit, Mich., OALR.

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Potter, Dr. Wm. Walter, Arnstein Bldg., Knoxville, Tenn., OALR.

Ramsey, Dr. G. Stuart, 374 Mountain St., Montreal, Can., Op.

Stitt, Dr. Howard Leigh, 656 Doctors Bldg., Cincinnati, Ohio. ALR.

Summers, Dr. Wm. Shakespeare, 3752 Vicksburg, Detroit, Mich., OALR.

Walker, Dr. Geo. H., Winona Clinic, Winona, Minn., OALR.

Woodworth, Dr. William P., 1424 Maccabees Bldg., Detroit, Mich., OALR.

Moved by the SECRETARY that the above candidates be elected to membership. Motion seconded and carried.

Moved by DR. ARTHUR J. BEDELL that a vote of thanks be extended to the local committee; also that a vote of thanks be extended to those mentioned by Dr. Parker in his report. Motion seconded and carried by rising vote.

DR. WALTER R PARKER, Detroit: I have been requested to make a short report of a meeting of the Commitee for the Revival of The International Congress of Ophthalmology.

The appointment of delegates from this country was made after correspondence with the officers in the leading national societies interested in ophthalmology, and Dr. de Schweinitz and I were named. The meeting was called at The Hague for July 12th. There were present forty-nine delegates from twenty-five nations. The meeting was held under the auspices of the Dutch ophthalmologists, and was in every respect as harmonious and satisfactory as possible.

I have not time to put before you the satisfaction and pleasure that came from this wonderful meeting. Imagine forty-nine delegates from twenty-five nations seated at one table, and at the end of the Conference in perfect accord, with The International Congress of Ophthalmology reestablished. The next Congress will be held at Amsterdam during the month of September, 1929. This country fared very well by having Dr. de Schweinitz made a member of the Council.

Considerable expense was incurred in connection with the meeting at The Hague. The Council felt that each of the national societies represented would perhaps be willing to make a contribution to help meet this expense. Persona!

contributions will also be very much appreciated, and the individual is requested to give not less than ten nor more than twenty-five dollars. Subscriptions are to be sent to Mr. Leslie Paton, 29 Harley St., London, W. 1.

THE PRESIDENT: Before adjournment I would like to say that the Secretaries, Dr. Wherry, Dr. Benedict, and Dr. Myers, have gone to no end of trouble in compiling this program. It was chosen from probably three times the number of papers that constitute the program. In other words, only about one-third of the papers offered were accepted. Of course the personal equation enters largely into what a man thinks is best on a program. I think most of us have been pretty well suited, but there is no question that the program could have been bettered. Probably each man here has an idea of something that he would like to hear discussed—and thinks some thing of little value could have been omitted. The thing I want to ask you to do is—when you go home, write a line to one of these secretaries—the one in whose specialty you are interested—as to what you would personally like next year. You have no idea how a few letters of this kind will help them in preparing the program for next year.

I might add that I have never seen a group of men who were better listeners than those attending this session, and on behalf of the Chair and the secretaries I want to thank you for the splendid cooperation you have given us in this program.

Moved by DR. R. H. T. MANN that a vote of thanks be extended to the men who were responsible for putting on this program. Motion seconded and carried. Adjourned.

ALPHABETIC LIST

Honorary Fellows

*Birkett, H. S. . . . . 1190 Mountain St., Montreal, Canada
Dundas-Grant, Sir James. . . 148 Harley St., W. 1, London, Eng.
Elliot, Lt.-Col. R. H.,
54 Welbeck St., Cavendish Sq., W., London, Eng.
Elschnig, Prof. A.,
Ferdinandstrasse, 10, Prague, Czecho-Slovakia
Graves, Basil. . . . . 35 Weymouth St., W., London, Eng.
Gray, Albert A. . . . . Glasgow, Scotland
Johnson, Lindsay,
Britannia Bldg., West St., Durban, Natal, Africa
Kirkpatrick, Lt.-Col. Henry,
54 Welbeck St., W. I., London, Eng.
Lawford, J. B., 27 Weymouth St., Portland Pl., W., London, Eng.
Lister, Sir William T., 24 Devonshire Place, W. 1, London, Eng.
Nager, Prof. F. . . . . Freiestrasse 20, Zurich, Switzerland
Van der Hoeve, Prof. J. . . Rijnsburgerweg, 6 A, Leiden, Holland

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Life Members

Brown, Edward J. (1911)
900 Nicollet Ave., Minneapolis, Minn., OALR
*Dayton, W. L. (1896) . . . 302 Funke Bldg., Lincoln, Neb., OALR
*Foster, Hal (1894)
1014 Chambers Bldg., Kansas City, Mo., ALR
Howe, Lucien. . . . . 520 Delaware Ave., Buffalo, N. Y., Op.
Lochard, L. B. . . . . Metropolitan Bldg., Denver, Colo.,
Mayer, Emil. . 666 West End Ave., New York City, N. Y., ALR
Posey, Wm. C. . . . . 2049 Chestnut St., Philadelphia, Pa.,
Standish, Myles,
51 Hereford St., Back Bay, Boston, Mass., Op.
Stewart, T. M. . . . . 901 Union Trust Bldg., Cincinnati, O., OALR
Wood, Casey A. . . . . 7 W. Madison St., Chicago, Ill.,

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*Attended 1927 meeting. Numbers in parentheses indicate year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary.

Fellows

A

ABBOTT, W. J. (1919) . . . . 8314 Euclid Ave., Cleveland, O., OALR
*ABRAHAMSON, IRA (1927)
605 Traction Bldg., Cincinnati, O., Op.
ADAMS, A. L. (1905) . . Professional Bldg., Jacksonville, Ill., OALR
ADAMS, C. F. . . . . 34 West State St., Trenton, N. J., OALR
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Moore Bldg., San Antonio, Tex., ALR
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122 Waterman St., Providence, R. I., ALR
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AGNEW, F. F. (1920) 120 3rd Ave., S. E., Independence, Ia., OALR
AINSLIE, GEORGE (1921)
606 Oregonian Bldg., Portland, Ore., OALR
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1st Brig. Marine, % Postmaster, New York City, N. Y., OALR
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537 Frisco Bldg., St. Louis, Mo., ALR
ALDERDYCE, WM. (1908) . . . 513 Madison Ave., Toledo, O., OALR
*ALEXANDER, GEO. J. . . . . 1629 Spruce St., Philadelphia, Pa., ALR
ALKIRE, H. L. (1898) . . . . . 614 Kansas Ave., Topeka, Kan., OALR
*ALLEN, CHAS. E. (1920)
712 Waldheim Bldg., Kansas City, Mo., OALR
ALLEN, G. H. (1916) . . . . . Mills Bldg., Topeka, Kan., OALR
ALLEN, G. V. (1921) . . . . . 835 Kansas Ave., Topeka, Kan., OALR
ALLEN, J. H. . . . . Republic Bldg., Denver, Colo., ALR
ALMOUR, R. W. (1925)
51 W. 73rd St., New York City, N. Y., ALR
ALTER, F. W. . . . . Toledo Medical Bldg., Toledo, O., OALR
ALTRINGER, A. N. (1920)
710 Rialto Bldg., Kansas City, Mo., ALR
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*AMBERG, EMIL. . . . . 662 Maccabee Bldg., Detroit, Mich., ALR
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ANDERSON, CARL M. (1926) Mayo Clinic, Rochester, Minn., ALR
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APPLEMAN, L. F. (1913)
308 South 16th St., Philadelphia, Pa., Op.
*ARBUCKLE, MILLARD F.,
1011 Beaumont Med. Bldg., St. Louis, Mo., ALR
ARNOLD, FRANCIS J. . . . . 182 Pearl St., Burlington, Vt., OALR

*ARRELL, W. . . . . .317 Main, E., Hamilton, Ontario, OALR
ARROWSMITH, HUBERT. . .170 Clinton St., Brooklyn, N. Y., ALR
*ASBURY, MARY K.(1925) . . . . .707 Race St., Cincinnati, O., Op.
ASH, GARRETT G.(1920) . . . . . .1 Main St., Bradford, Pa., OALR
ASHLEY, T. W. . . . . .5711 8th Ave., Kenosha, Wis., OALR
ATKINSON, D. T.(1918)
827 Med. Arts Bldg., San Antonio, Tex., OALR
*ATKINSON, WALTER S.(1925)
168 Sterling St., Watertown, N. Y., Op.
AUERBACH, JULIUS.120 W. 86th St., New York City, N. Y., ALR
AYNESWORTH, H. T.(1912)
1307 Amicable Bldg., Waco, Tex., OALR

B

BABBITT, JAMES A.(1924)
1912 Spruce St., Philadelphia, Pa., ALR
*BAHN, CHAS. A.(1922)
1707 Pere Marquette Bldg., New Orleans, La., Op.
BAILEY, FRED W.(1912) Security Bldg., Cedar Rapids, Ia., OALR
BAILEY, HAROLD(1908)
840 Landers Bldg., Springfield, Mo., OALR
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*BAKER, CHAS. H. . . . . .Crapo Blk., Bay City, Mich., OALR
BAKER, HARRY B.(1916) . . . . .59 Main St., Taunton, Mass., OALR
BALDWIN, KATE W. . . . .1117 Spruce St., Philadelphia, Pa., ALR
BALENTINE, P. L.(1915)
1524 Chestnut St., Philadelphia, Pa., Op.
BALL, JAMES MOORES. . . . .4500 Olive St., St. Louis, Mo., Op.
BALL, MICHAEL V. . . . . .316 Hazel St., Warren, Pa., OALR
BALLENGER, H. C(1915)
25 E. Washington St., Chicago, Ill., ALR
BALLIN, M. J. . . . . .108 E. 60th St., New York City, N. Y., ALR
BALLON, D. H.(1924) . .107 Crescent St., Montreal, Canada, ALR
BANE, WM. C. . . . . .1005 Republic Bldg., Denver, Colo., OALR
BANE, W. M. . . . . .1005 Republic Bldg., Denver, Colo., OALR
*BANISTER, COL. J. M.(1913)
Bankers Reserve Life Bldg., Omaha, Neb., OALR
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BARCK, CARL(1896) . . . . . .Humboldt Bldg., St. Louis, Mo., Op.A
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BARKAN, ADOLPH . .Union Trust Bldg., San Francisco, Cal., OALR
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BARKER, O. G. A.(1909)
804 Johnstown Trust Co., Johnstown, Pa., Op.
BARLOW, ROY A. . . . . .Madison, Wis., ALR
BARNES, L. E.(1914) 6860 South Halsted St., Chicago, Ill., OALR
*BARNHILL, J. F.(1896)
408 Pennway Bldg., Indianapolis, Ind., ALR

BARTHOLOMEW, A. C.(1904)
1020 Harrison St., Ft. Wayne, Ind., OALR
*BASS, A. L.(1924).....738 Francis Bldg., Louisville, Ky., OALR
BAUM, H. L.(1917)....510 Republic Bldg., Denver, Colo., ALR
BEACH, B. S.(1927).....76 W. 86th St., New York, N.Y., Op.
*BEACH, S. J.(1914).....704 Congress St., Portland, Me., Op.
BEAL, HOMER A.(1926)
1002 Argyle Bldg., Kansas City, Mo., ALR
*BEARD, H. J.(1924)
510 Dollar Bank Bldg., Youngstown, O., OALR
*BEATTIE, ROBERT(1906)
David Whitney Bldg., Detroit, Mich., Op.
BEATTIE, WILLIAM H.(1921) 252 Genesee St., Utica, N. Y., OALR
BEATTY, H. G.....681 E. Broad St., Columbus, O., OALR
BECK, A.L.(1927).421 Huguenot St., New Rochelle, N. Y., OALR
BECK, FREDERICK L.(1920)
408 Hynds Bldg., Cheyenne, Wyo., OALR
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BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
*BEDELL, ARTHUR J.....344 State St., Albany, N. Y., Op.
*BEEBE, C. S.....509 Wells Bldg., Milwaukee, Wis., Op.
*BEEM, IONE F.(1916).....31 N. State St., Chicago, Ill., OALR
BEGGS, W. F.(1914).....2 Lombardy St., Newark, N. J., OALR
BEIL, J. W.(1918).....Argyle Bldg., Kansas City, Mo., OALR
BELL, M. H.(1913)
First Nat'l Bank Bldg., Vicksburg, Miss., Op.
BELLIN, JOSEPH.....1052 W. 6th St., Los Angeles, Cal., OALR
BELLOWS, GEORGE E.(1896)
Rialto Bldg., Kansas City, Mo., Op.
*BENEDICT, WILLIAM L.(1919)
Mayo Clinic, Rochester, Minn., Op.
BENNETT, ARTHUR G...150 N. Pearl St., Buffalo, N. Y., Op.
*BENTLEY, NEIL, 1161 David Whitney Bldg., Detroit, Mich., OALR
*BERENS, CONRAD(1924)
30 E. 40th St., New York City, N. Y., Op.
BERGERON, J. Z.....104 S. Michigan Blvd., Chicago, Ill., ALR
BERNATZ, C. F.....Park Bldg., Pittsburgh, Pa., Op.
*BERNSTEIN, E. J(1900).....10 Peterboro, Detroit, Mich., OALR
BERRISFORD, PAUL D.....Lowry Bldg., St. Paul, Minn., OALR
*BERRY, D. F.(1911)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
BETTISON, D. L.....1102 Medical Arts Bldg., Dallas, Tex., OALR
*BEYER, A. G.(1925).....19 Garfield Pl., Cincinnati, O., OALR
BEYER, LOUIS J.(1918) 449 Delaware Ave., Buffalo, N. Y., ALR
BINGER, H. E.(1917)....824 Lowry Bldg., St. Paul, Minn., OALR
*BINKLEY, ROY S.(1915)....530 Fidelity Bldg., Dayton, O., OALR
*BIRSNER, LOUIS J.(1926)
25 1st Nat'l Bk Bldg., Belleville, Ill., ALR
*BLAAUW, E. E.....190 Ashland Ave., Buffalo, N. Y., Op.
*BLACK, MELVILLE(1904)
424 Metropolitan Bldg., Denver, Colo., Op.

*Black, N. M.....1240 Wells Bldg., Milwaukee, Wis., Op.
Black, Paul (1927).514 1st Natl. Bk Bldg., Lincoln, Neb., OALR
Black, W. D. (1900)..Metropolitan Bldg., St. Louis, Mo., ALR
*Blackburn, W. J. (1919)..661 Reibold Bldg., Dayton, O., OALR
Blair, W. W.....Diamond Bank Bldg., Pittsburgh, Pa., Op.
Blake, E. M.....121 Whitney Ave., New Haven, Conn., Op.
*Blake, Wm. F.....490 Post St., San Francisco, Cal., Op.
*Blakesly, T. S.....Lathrop Bldg., Kansas City, Mo., OALR
Blassingame, C. D. (1924)
414 Phys. and Surg. Bldg., Memphis, Tenn., ALR
Bledsoe, R. W. (1908) 1005 Madison Ave., Covington, Ky., OALR
*Blickenstaff, A. J. (1922)
623 Peoria Life Bldg., Peoria, Ill., OALR
Bliss, C. B.....411 Columbus Ave., Sandusky, O., OALR
Blue, J. B. (1916)....Columbian Tower, Memphis, Tenn., OALR
Blum, Henry
1211 Maison Blanche Bldg., New Orleans, La., Op.
Bobb, E. V. (1918).....Mitchell, S. D., OALR
Boeckmann, Egil.....Lowry Bldg., St. Paul, Minn., OALR
Boehringer, H. W,
1823 Spring Garden St., Philadelphia, Pa., Op.
Boerner, Morris H. (1923)
Scarborough Bldg., Austin, Tex., OALR
*Boiler, W. F. (1912).....Iowa City, Ia., OALR
Bone, Herman D.....19 Pleasant St., Gardner, Mass., OALR
Bonner, Wm. F. (1926)
224 Del. Tr. Bldg., Wilmington, Del.
*Boone, J. C. (1921)..213 Pythian Bldg., South Bend, Ind., ALR
*Boone, J. F (1927)...30 N. Michigan Blvd., Chicago, Ill., OALR
Boone, J. L. (1924)..Professional Bldg., Jacksonville, Fla., ALR
Boot, G. W. (1909).....813 Sherman Ave., Evanston, Ill., OALR
Bordley, James, Jr...330 N. Charles St., Baltimore, Md., OALR
Boucher, R. B.....Birks Bldg., Vancouver, B. C., OALR
Bourbon, Oliver P. (1920)
607 S. Hill St., Los Angeles, Cal., OALR
Bousquet, J. P. E. (1924)
26 Sherbrooke St., W., Montreal, Can., OALR
Bouvy, Harry (1920)....800 Pittock Blk., Portland, Ore, OALR
Bouvy, L. B. (1927).....La Grande, Ore., OALR
*Boyce, G. H. (1912)..408 E. D. St., Iron Mountain, Mich., OALR
*Boyce, W. B. (1912).1107 Ludington St., Escanaba, Mich., OALR
Boyd, E. T. (1896).....823 Majestic Bldg., Denver, Colo., OALR
Boyd-Snee, Harry, 716 J. M. S. Bldg., South Bend, Ind., ALR
Bozer, Herrmann Eugene (1923)
438 Delaware Ave., Buffalo, N. Y., ALR
Bradfield, J. A. L.....1515 Main St., La Crosse, Wis., OALR
Braswell, James C. (1922).708 Mayo Bldg., Tulsa, Okla., OALR
Braunlin, Wm. H. (1920)
Marion Natl. Bank Bldg., Marion, Ind., OALR
Brawley, F. E.....30 N. Michigan Blvd., Chicago, Ill., Op.
Brawley, R. V.....Salisbury, N. C., Op.

BRAY, E. R.(1912).....342 Lowry Bldg., St. Paul, Minn., OALR
BREHMER, HARRISON L.(1920)
1st Natl. Bk. Bldg., Albuquerque, N. M., OALR
BREWER, JOHN D.(1920).....Dyersburg, Tenn., OALR
BRIBACH, EUGENE J.(1919)
212 Simpson Bldg., Atchison, Kan., OALR
*BRICKLEY, DANIEL W.(1918)
196 W. Center St., Marion, O., OALR
BRIGGS, F. W.....Fidelity Bldg., Duluth, Minn., OALR
*BRIGGS, H. H.....101 Haywood St., Asheville, N. C., OALR
BRIGGS, W. E.....1027 10th St., Sacramento, Cal., OALR
BRITTEN, G. S.(1907)....University Blk., Syracuse, N. Y., ALR
*BROBST, C. H.(1907).....Central Bank Bldg., Peoria, Ill., OALR
*BRODRICK, FRANK W....511 Lawrence Bldg., Sterling, Ill., OALR
*BROOKS, EARL B.(1921)
707 1st Natl. Bk. Bldg., Lincoln, Neb., OALR
BROOKS, E. H.(1915).108 W. College Ave., Appleton, Wis., OALR
BROSE, L. D.....501 Upper First St., Evansville, Ind., OALR
BROWN, ALBERT LOUIS(1926)
2600 Union Central Bldg., Cincinnati, O., Op.
*BROWN, A. O.(1927)..742 Maccabee Bldg., Detroit, Mich., OALR
BROWN, C. M.....510 Delaware Ave., Buffalo, N. Y., ALR
*BROWN, C. W.(1927)
613 Commonwealth Bldg., San Diego, Cal., ALR
BROWN, EDMUND TOWLE...30 Church St., Burlington, Vt., OALR
BROWN, H. ALEXANDER(1912)
291 Geary St., San Francisco, Cal., Op.
BROWN, JOHN C.(1920) 632 Lowry Bldg., St. Paul, Minn., OALR
BROWN, JOHN E.(1901) 370 East Town St., Columbus, O., OALR
BROWN, J. M.....1136 W. 6th St., Los Angeles, Cal., ALR
BROWN, L. E.(1917)....2nd Natl. Bank Bldg., Akron, O., OALR
BROWN, MORROW D....432 Republic Bldg., Denver, Colo., ALR
*BROWN, MORTIMER G.(1921)
802 University Bldg., Syracuse, N. Y., ALR
BROWN, SAMUEL HORTON(1910)
1901 Mt. Vernon St., Philadelphia, Pa., Op.
*BROWN, W. HAROLD(1912)
Bk. of Montreal Bldg., Edmonton, Alberta, Can., OALR
BRUDER, J.(1908)..229 W. 78th St., New York City, N. Y., OALR
BRUERE, G. E.(1919).....Journal Bldg., Portland, Ore., OALR
BRUMM, SETH A.(1922)
818 Stock Exchange Bldg., Philadelphia, Pa., ALR
BRUNER, ABRAM BRENEMAN(1922)
1214 Guardian Bldg., Cleveland, O., Op.
BRUNER, WM. E.....Guardian Bldg., Cleveland, O., Op.
BRUST, H. O.(1913)..720 S. Crouse Ave., Syracuse, N. Y., ALR
BRYANT, ALICE G.(1908)...502 Beacon St., Boston, Mass., ALR
BUCHANAN, NORMAN D.(1925)
479 Water St., Peterborough, Ont., Canada, OALR

BUCKLEY, J. H.(1908).1st Natl. Bk. Bldg., Ft. Smith, Ark., OALR
BUCKMAN, E. U.(1906)
70 S. Franklin St., Wilkes-Barre, Pa., OALR
BULL, EDWARD L.....Savings Bk. Bldg., Ithaca, N. Y., OALR
*BULSON, A. E., JR.(1896)
406 W. Berry St., Ft. Wayne, Ind., OALR
*BURCH, FRANK E.(1907) 754 Linwood Pl., St. Paul, Minn., Op.
*BURDICK, AUSTIN F.(1925)
416 Tussing Blk., Lansing, Mich., OALR
BURKE, JOHN R.(1927).131 Chestnut St., Holyoke, Mass., OALR
BURKE, T. A.....Rose Bldg., Cleveland, O., OALR
BURLESON, J. H.(1917)
Eye, Ear, Nose and Throat Hospital, San Antonio, Tex., OALR
BURNS, S. S.(1919).....Humboldt Bldg., St. Louis, Mo., ALR
BURROUGHS, G. M.(1911)..5 Broad St., Danielson, Conn., Op.
BURTON, FRANK A.(1914)...Watts Bldg., San Diego, Cal., ALR
*BUSHMAN, L. B.(1927)
631 City Natl. Bk. Bldg., Omaha, Neb., OALR
BUTLER, MARGARET F., 1831 Chestnut St., Philadelphia, Pa., ALR
BUTLER, ROBERT H.(1920).....Bellefontaine, O., OALR
BUTT, MIRIAM M.....2045 Chestnut St., Philadelphia, Pa., Op.
BUVINGER, CHARLES W.(1921)
50 Washington St., East Orange, N. J., OALR
BYRNES, H. F.....67 Chestnut St., Springfield, Mass., OALR

C

*CAHILL, J. W.....390 Main St., Worcester, Mass., OALR
CALDWELL, ROBERT
831 New Donaghey Bldg., Little Rock, Ark., OALR
*CALHOUN, F. PHINIZY(1911)
436 Peach Tree St., Atlanta, Ga., Op.
CALLFAS, W. F.....1620 Med. Arts Bldg., Omaha, Neb., ALR
CAMERON, WM. G...Puget Sd. Bk. Bldg., Tacoma, Wash., OALR
*CAMP, W. E.(1920)
716 Donaldson Bldg., Minneapolis, Minn., OALR
CAMPBELL, C. H.....2904 15th St., Columbus, Neb., OALR
*CAMPBELL, D. A.....David Whitney Bldg., Detroit, Mich., OALR
*CAMPBELL, DON M.(1908)
David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, M. D.(1927)
David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, ROBERT A.,
520 LaSalle Bldg., Minneapolis, Minn., OALR
*CAMPBELL, W. K.....96 Third Ave., Long Branch, N. J., OALR
CAPRON, F. P.....118 Angell St., Providence, R. I., OALR
CARLETON, RALPH(1914) 10 Temple St., Springfield, Mass., Op.
CARMACK, J. W.(1919)
505 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CARMODY, T. E.(1904)..Metropolitan Bldg., Denver, Colo., ALR

*CARMODY, THOMAS J. (1926)
408 The Temple, Danville, Ill., ALR
CARNCROSS, HELEN (1920)
58 E. Washington St., Chicago, Ill., Op.
CARPENTER, E. R. . . . . 708 Medical Arts Bldg., Dallas, Tex., OALR
CARPENTER, E. W. (1920) . . . Prof. Bldg., Greenville, S. C., OALR
CARR, GEO. W. (1909) 54 S. Franklin St., Wilkes-Barre, Pa., OALR
CARROLL, GEO. G. . . . . 614 W. Main St., Rochester, N. Y., OALR
*CARSON, W. E. . . . . Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
CARTER, W. W. (1896) 2 E. 54th St., New York City, N. Y., ALR
CARVILLE, L. MAUD (1914)
82 Commonwealth Ave., Boston, Mass., Op.
CARY, E. H. . . . . 4712 Lake Side Drive, Dallas, Tex., OALR
CARY, G. C. . . . . Grand Junction, Colo., OALR
CAVANAUGH, J. A. . . . 1712-30 N. Mich. Blvd., Chicago, Ill., ALR
*CAYCE, E. B. (1912) . . . . Med. Arts Bldg., Nashville, Tenn., OALR
*CHADWICK, IRA B. (1920)
4 Terminal Bldg., Coffeyville, Kan., Op.
CHAPMAN, SOLOMON J. (1925)
402 Burns Bldg., Colorado Springs, Colo., ALR
CHAPMAN, V. A. . . . Colby-Abbott Bldg., Milwaukee, Wis., OALR
*CHAPPELL, R. S. (1925)
305 Terminal Bldg., Indianapolis, Ind., ALR
CHARLES, JOS. W. . . . . 537 N. Grand Ave., St. Louis, Mo., Op.
CHARLTON, C. C. . 124 S. Illinois Ave., Atlantic City, N. J., ALR
CHASE, E. FRANK (1918) 1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, S. B. (1925) . . . . . Carver Bldg., Ft. Dodge, Ia., OALR
*CHASE, W. D. (1924) . . . . 230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. F. . . . . 671 Broad St., Newark, N. J., Op.
CHENERY, W. E. . 377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, JAMES W. . . 702 Schweiter Bldg., Wichita, Kan., OALR
*CHILDS, H. A. . . . . Eagle Blk., Creston, Ia., OALR
CHIPMAN, L. D. V. (1925)
42 Coburg St., St. John, N. B., Canada, OALR
*CLARK, H. S. (1916) . . . LaSalle Bldg., Minneapolis, Minn., OALR
*CLARK, I. G. . . . . 186 E. State St., Columbus, O., OALR
*CLARK, J. SHELDON (1914)
509 State Bk. Bldg., Freeport, Ill., OALR
CLARK, L. H. (1915) . . 337 Monroe Ave., Rochester, N. Y., OALR
*CLAY, G. E. (1917) . . . . . Med. Arts Bldg., Atlanta, Ga., Op.
*CLEFF, OSCAR (1926) . . . . . 31 N. State St., Chicago, Ill., OALR
CLEMENT, C. C. . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR
CLERF, LOUIS HENRY (1923)
128 S. 10th St., Philadelphia, Pa., ALR
*CLEVENGER, W. F. (1905)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CLIFFORD, T. P. (1927)
1320 David Whitney Bldg., Detroit, Mich., OALR
CLOTHIER, JOSEPH . . . . . Pocatello, Ida.
*COATES, GEO. M. (1922) . . . 1721 Pine St., Philadelphia, Pa., ALR

COBB, EDWIN (1912) . . Masonic Temple, Marshalltown, Ia., OALR
COFFELT, THEODORE A.
508 Woodruff Bldg., Springfield, Mo., OALR
COGAN, J. E. . . . . 707 Rose Bldg., Cleveland, O., OALR
COHEN, HERMAN B. (1927)
Spruce & 13th St., Philadelphia, Pa , ALR
COHEN, MARTIN, 1 West 85th St., New York City, N. Y., Op.
COHEN, SAMUEL (1920) . 1626 Spruce St., Philadelphia, Pa., ALR
COHN, FELIX . . . . . 47 W. 87th St., New York City, N. Y., ALR
COHN, SELDON . . . . . Fulton, Ky., OALR
COLLINS, E. W. . . . . 508 Majestic Bldg., Denver, Colo., ALR
COLLINS, J. D. (1914) . . 150 Main St., Northampton, Mass., ALR
COLVER, B. N. (1913)
182 Manchester St., Battle Creek, Mich., ALR
COMEE, W. C. (1922) . . . . . Glenwood Springs, Colo., OALR
CONLON, F. A. . . . . Bay State Bldg., Lawrence, Mass., OALR
*CONNOR, CHAS. E. (1920) . . . Hamm Bldg., St. Paul, Minn., ALR
*CONNOR, RAY (1905) . . . . . 1410 Stroh Bldg., Detroit, Mich., OALR
COOK, W. ALBERT (1924) . . . 506 Palace Bldg., Tulsa, Okla., OALR
*COOPER, C. E. . . . . 652 Metropolitan Bldg., Denver, Colo., ALR
COOVER, D. H. (1918)
820 Metropolitan Bldg., Denver, Colo., Op.
COPPS, LYMAN ALDEN (1923) . . . . . Marshfield, Wis., OALR
CORBETT, J. J. (1925) . . . . . 520 Beacon St., Boston, Mass., OALR
*CORSER, J. B. . . . . 345 Wyoming Ave., Scranton, Pa., Op.
*COSTEN, J. B. (1925) 3720 Washington Ave., St. Louis, Mo., ALR
*COTT, C. C. . . . . 483 Delaware Ave., Buffalo, N. Y., ALR
COTT, D. N. (1924)
141 St. Marks Pl., St. George, Staten Isl., New York, ALR
COULTER, CHAS. F.
404 1st Natl. Bk. Bldg., Great Falls, Mont., OALR
*COWLEY, ROBERT HENRY (1926) . . . . . Berea, Ky., OALR
*COWPER, H. W. . . . . 543 Franklin St., Buffalo, N. Y., Op.
CRAFT, KENNETH L. (1920)
1003 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CRANE, J. WELLINGTON (1921)
128 Third Ave., Trenton, N. J., OALR
CREIGHTON, WM. J. (1910)
258 S. 18th St., Philadelphia, Pa., Op.
*CRISP, WM. H. (1913) . . . . Metropolitan Bldg., Denver, Col., Op.
CROCKETT, R. L. . . . . 302 Main St., Oneida, N. Y., Op.
CROMAN, JOS. M. JR. (1924) . . . . . Mount Clemens, Mich., OALR
CROSKEY, J. W. (1905) . . . . Aldine Hotel, Philadelphia, Pa., Op.
*CROSS, A. E. (1914) . . . . . 390 Main St., Worcester, Mass., Op.
CROSS, G. H. (1919) . . . . . 525 Welsh St., Chester, Pa., Op.
CROSSLEY, E. R. . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR
CULBERTSON, L. R. (1898) Masonic Temple, Zanesville, O., OALR
CULLOM, M. M. (1910)
1119 Bennie-Dillon Bldg., Nashville, Tenn., OALR

CUNNINGHAM, ALLAN R.(1924)
260 Barrington St., Halifax, N. S., OALR
CURDY, R. J.....1134 Rialto Bldg., Kansas City, Mo., Op.
CURRAN, E. J.(1919)
1805 Federal Reserve Bk. Bldg., Kansas City, Mo., Op.
CURRY, GLENDON E.(1912)
Westinghouse Bldg., Pittsburgh, Pa., Op.
CURTIN, EUGENE A.(1923)
703 Connell Bldg., Scranton, Pa., OALR
CURTIS, W. L.....1503 So. 22nd St., Lincoln, Neb., OALR
*CUSHMAN, BEULAH(1924)
25 E. Washington St., Chicago, Ill., Op.
CUTLER, FRANKLIN E..1026 Schofield Bldg., Cleveland, O., ALR

D

DABNEY, S. G.....Starks Bldg., Louisville, Ky., OALR
DABNEY, VIRGINUS(1913)
1633 Connecticut St., Washington, D. C., ALR
*DAILY, LOUIS(1926) 1117 Med. Arts Bldg., Houston, Tex., OALR
*DAILY, RAY K.(1926)
1117 Med. Arts Bldg., Houston, Tex., OALR
*DANCY, ALEXANDER B.
First Natl. Bank Bldg., Jackson, Tenn., OALR
DANIELS, R. L.....New Bern, N. C., OALR
DARMER, GEO. A.(1921)..401 Terminal Bldg., Aurora, Ill., OALR
DAVIDSON, H. P.(1924) 58 E. Washington St., Chicago, Ill., Op.
*DAVIES, J. S.(1927) 704 St. Helens Ave., Tacoma, Wash., OALR
*DAVIES, T. A.(1913)....578 Sherborne St., Toronto, Can., OALR
DAVIS, A. EDWARD..47 E. 57th St., New York City, N. Y., Op.
*DAVIS, F. A.(1927)
122 W. Washington Ave., Madison, Wis., OALR
DAVIS, G. E.....42 W. 77th St., New York City, N. Y., OALR
*DAVIS, HARRY B.(1920)
712 Bryant Bldg., Kansas City, Mo., OALR
DAVIS, J. E.....Kress Bldg., McAlester, Okla., OALR
DAVIS, J. L.....135 S. 18th St., Philadelphia, Pa., ALR
DAVIS, RALPH F.(1920) 1104 Selling Bldg., Portland, Ore., OALR
DAVIS, THOMAS W.,
210 O'Hanlon Bldg., Winston-Salem, N. C., OALR
DAVIS, W. T.(1919)..927 Farragut Sq., Washington, D. C., Op.
DAYTON, G. O.....245 Bradbury Bldg., Los Angeles, Cal., OALR
DEAN, ALFRED....304 Gilbert Bldg., Grand Rapids, Mich., Op.
*DEAN, F. W.(1904)
Council Bluffs Clinic, Council Bluffs, Ia., OALR
DEAN, L. W.....12 S. Clinton St., Iowa City, Ia., ALR
*DEAN, WALTER(1917)..300 Frances Bldg., Louisville, Ky., OALR
DEBOE, M. P.....1st Natl. Bk. Bldg., Miami, Fla., OALR
DECHERD, H. B.....1515 Med. Arts Bldg., Dallas, Tex., OALR
*DECKER, J. C.(1918).....Francis Bldg., Sioux City, Ia., OALR

*DEFNET, WM. A.(1921)..1309 Stroh Bldg., Detroit, Mich., ALR
DEICHLER, LYN W...1737 Chestnut St., Philadelphia, Pa., OALR
DENMAN, IRA O.(1925).....425 Ohio Bldg., Toledo, O., OALR
*DENNIS, DAVID N.(1902)....221 W. Ninth St., Erie, Pa., Op.
*DENNIS, EDWARD P.(1921)....309 W. 10th St., Erie, Pa., OALR
DENNIS, F. L.(1906)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
*DENNIS, GEO. J.(1910).25 E. Washington St., Chicago, Ill., ALR
DERBY, GEO. S.....23 Bay State Rd., Boston, Mass., Op.
DETLING, FRANK E..907 Chapman Bldg., Los Angeles, Cal., OALR
DERIVER, J. P.(1919)
Veterans Bureau, San Francisco, Cal., OALR
DEWEESE, CLARENCE...154 N. Upper St., Lexington, Ky., OALR
DEWEY, C. H.(1912)
Sternberg General Hospital, Manilla, P. I.
DICKINSON, B. M.
5634 Stanton Ave., E. Liberty Sta., Pittsburgh, Pa., ALR
DINTENFASS, HENRY....1305 Spruce St., Philadelphia, Pa., ALR
DIXON, G. S.....218 2nd Ave., New York City, N. Y., R
DIXON, W. E..503 Med. Arts Bldg., Oklahoma City, Okla., OALR
DOANE, L. L.(1905).....357 N. Main St., Butler, Pa., OALR
DODD, OSCAR(1903).....708 Church St., Evanston, Ill., Op.
DOHERTY, WM. B.(1927)
150 W. 55th St., New York City, N. Y., Op.
DOLMAN, PERCIVAL(1919)
490 Post St., San Francisco, Cal., Op.
DONNELL, N. R.(1917) Metropolitan Bldg., St. Louis, Mo., Op.
DONOHER, WM. D....Boston Bldg., Salt Lake City, Utah., OALR
DONOVAN, JNO. A.(1902) 507 Phoenix Bldg., Butte, Mont., OALR
*DORENTE, D. R....1st Natl. Bank Bldg., Fort Smith, Ark., OALR
DORGE, RICHARD IRVING(1922)
76 Seymour Ave., S. E., Minneapolis, Minn., OALR
DORSEY, J. G.....201 N. Main St., Wichita, Kan., OALR
DOUGLAS, FREDERICK ALEXANDER(1922)
303 State Bk. Bldg., La Crosse, Wis., OALR
DOW, FRANK E.(1914)..4 West St., Northampton, Mass., Op.
DOWLING, J. IVIMEY.116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. T.(1915)
Virginia Mason Hosp. Bldg., Seattle, Wash., OALR
*DUANE, J. F.(1916).....Jefferson Bldg., Peoria, Ill., OALR
DUDLEY, W. H.(1902)..520 W. 7th St., Los Angeles, Cal., OALR
DULANEY, OCTAVUS.....811 Brown Bldg., Louisville, Ky., ALR
DUNLAP, LAWRENCE G.(1920)
Electric Light Bldg., Anaconda, Mont., OALR
*DUNN, MURISON.....Richmond, Ky., OALR
DUNN, O. B.....4th and Railroad Sts., Ironton, O.
*DUNNINGTON, J. H.(1924)
30 W. 59th St., New York City, N. Y., Op.
DUNTLEY, G. S.(1913).....103 So. Side Sq., Macomb, Ill., OALR

DURR, S. A.(1927)
1304 Medico-Dental Bldg., San Diego, Cal., Op.
*DUTROW, H. V.(1914)....1040 Fidelity Bldg., Dayton, O., OALR
DYER, C. P.(1920) 233 Metropolitan Bldg., St. Louis, Mo., Op.

E

*EAREL, A. M.(1912)...507 Willdon Bldg., Hoopeston, Ill., OALR
EASTON, J. C.(1904).....Fairbanks Bldg., Springfield, O., OALR
EDSON, R.....498 Delaware Ave., Buffalo, N. Y., Op.
*EDWARDS, C. J.(1913)
First National Bank Bldg., Vicksburg, Miss., OALR
EDWARDS, SLOCOMB R.(1918)
Christman Bldg., South Bend, Ind., OALR
ELLEGOOD, J. A....408 Equitable Bldg., Wilmington, Del., OALR
*ELLETT, E. C.(1898)...Exchange Bldg., Memphis, Tenn., Op.
ELLIS, Z. H.(1927) 30 W. 59th St., New York City, N. Y., Op.
*ELWOOD, CALVIN R.(1922).....Menominee, Mich., OALR
*EMBERY, FRANK(1927)
4660 Frankford Ave., Philadelphia, Pa., ALR
EMERSON, LINN(1906) Metropolitan Bldg., Orange, N. J., OALR
ENGLE, H. P.....200 Olive St., Newton, Ia., OALR
*ERSNER, MATTHEW S...1915 Spruce St., Philadelphia, Pa., ALR
*ESTABROOK, CHAS. T...857 Pleasant St., Worcester, Mass., OALR
*EVANS, J. N.(1925) 23 Schermerhorn St., Brooklyn, N. Y., Op.
EWING, ARTHUR E....Metropolitan Bldg., St. Louis, Mo., Op.
*EZELL, H. C.(1926)..228 Doctors Bldg., Nashville, Tenn., OALR

F

FAIRBAIRN, JOHN.....503 Delaware Ave., Buffalo, N. Y., ALR
FAIRCHILD, NORA M.(1922)
Med. Arts Bldg., Omaha, Neb., Op.
FAIRING, J. W.(1924)
205 Tribune Review Bldg., Greensburg, Pa., OALR
*FAITH, THOMAS.....31 N. State St., Chicago, Ill., OALR
*FALLOWS, HOWARD D.(1916)
Park Hospital Clinic, Mason City, Ia., OALR
FARMER, A. G.(1919)....Fidelity Med. Bldg., Dayton, O., OALR
FARNSLER, H. HERSHEY..1438 Market St., Harrisburg, Pa., OALR
FARNSWORTH, M. A.(1917)..2436–6th St., Boulder, Colo., OALR
FARRELL, P. J. H.(1902)
25 E. Washington St., Chicago, Ill., OALR
FARRINGTON, P. M.(1904)
Exchange Bldg., Memphis, Tenn., ALR
*FAWCETT, I.(1913).....75–12th St., Wheeling, W. Va., OALR
FELLOWS, C. G.....30 N. Michigan Blvd., Chicago, Ill., OALR
FELT, C. L.....Chestnut & 20th Sts., Philadelphia, Pa., ALR
*FENTON, RALPH A.(1921)
806 Medical Arts Bldg., Portland, Ore., OALR
FERGUSON, E. S.(1906)
902 Medical Arts Bldg., Oklahoma City, Okla., OALR

*FERGUSON, R. J.(1927)
59 College St., New Haven, Conn., OALR
*FERMAN, JOHN W.(1921)..Stahlman Bldg., Charleroi, Pa., OALR
FIELDING, F. G.(1925)....72 Ridge St., Glens Falls, N. Y., ALR
FIGI, F. A.(1927).....Mayo Clinic, Rochester, Minn., ALR
FINK, WALTER H.(1927)
426 LaSalle Bldg., Minneapolis, Minn., OALR
*FINNOFF, W. C.(1916)..217 Imperial Bldg., Denver, Colo., Op.
FISCHER, N. A.(1925)
623 Union Trust Bldg., Pittsburgh, Pa., ALR
FISCHER, V. B..230 Mercantile Bk. Bldg., Boulder, Colo., OALR
FISHER, CARL.....538 W. 6th St., Los Angeles, Cal., Op.
*FISHER, JAMES A.(1925).Fitkin Bldg., Asbury Park, N. J., OALR
FISHER, W. A.....31 N. State St., Chicago, Ill., OALR
FISKE, GEO. F.....25 E. Washington St., Chicago, Ill., OALR
FITZGERALD, J. P.(1924)
104 S. Michigan Blvd., Chicago, Ill., Op.
FLAGG, J. D.(1900).....473 Virginia Ave., Buffalo, N. Y., Op.A
*FLATLEY, T. J.(1914).....Reliance Bldg., Moline, Ill., OALR
FLOYD, T. W.(1918)...811 Peoria Life Bldg., Peoria, Ill., OALR
FLYNN, CHAS. T.(1927)
1138 Chapel St., New Haven, Conn., ALR
FLYNN, JAMES A.(1924)
1511 Rhode Island Ave., N. W., Washington, D. C., ALR
FOGARTY, CHAS. W.(1920)
1826 Summit Ave., St. Paul, Minn., OALR
FOLEY, J. W.(1915).....Majestic Bldg., Denver, Colo., OALR
*FOLSOM, W. H.(1927)
395 E. Division St., Fond du Lac, Wis., OALR
*FORBES, SHERMAN B.(1921)
409 Citizens Bk. Bldg., Tampa, Fla., OALR
*FORGRAVE, L. ROBERT(1922)
204 Logan Bldg., St. Joseph, Mo., Op.
*FORSYTHE, S. T.(1919) 235 Colonial Arcade, Cleveland, O., OALR
FORT, A. G.....1729 Decatur Rd., Atlanta, Ga., OALR
FOSTER, J. H.....1306 Walker Ave., Houston, Tex., ALR
FOSTER, J. McE.....Metropolitan Bldg., Denver, Colo., OALR
*FOUTS, JOHN D.(1920).....910 Fidelity Bldg., Dayton, O., OALR
FOWLER, EARL B.(1925).....31 N. State St., Chicago, Ill., Op.
FOWLER, S. R.....University Bldg., Syracuse, N. Y., ALR
*FOWLER, WM.(1925) 1048 Maccabee Bldg., Detroit, Mich., OALR
*FOX, C. H.(1920).....2119 Central Ave., Kearney, Neb., OALR
FOX, L. WEBSTER.....303 S. 17th St., Philadelphia, Pa., Op.
FOX, NOAH(1926)..6306 Cottage Grove Ave., Chicago, Ill., ALR
*FOX, RALPH D.(1916).....Unity Bldg., Bloomington, Ill., OALR
FRANCHERE, FRED'K E.(1920)
4th and Nebraska Sts., Sioux City, Ia., OALR
FRANK, IRA(1908)....104 S. Michigan Blvd., Chicago, Ill., ALR
*FRASER, R. H.(1925)
Battle Creek Sanitarium, Battle Creek, Mich., ALR

*Fraser, W.G.(1927).....52 Nepean St., Ottawa, Canada, OALR
Freedman, L. M.(1920)
475 Commonwealth Ave., Boston, Mass., ALR
Freimuth, L. E.(1927)
234 University Club Bldg., St. Louis, Mo., ALR
*French, R. F.(1913) Masonic Temple, Marshalltown, Ia., OALR
Freudenthal, Wolff
24 W. 88th St., New York City, N. Y., ALR
Frey, Clarence L.....529 Vine St., Scranton, Pa., OALR
Friedenwald, Harry....1212 Eutaw Pl., Baltimore, Md., Op.A
Friedenwald, J. S.(1925) 1212 Eutaw Pl., Baltimore, Md., Op.
*Fringer, W. R.....William Brown Bldg., Rockford, Ill., Op.
Fulkerson, C. B.(1914)
308 Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
Fullenwider, C. M..404 Barnes Bldg., Muskogee, Okla., OALR
Fuller, Harry B.(1925)...2219 E 70th Pl., Chicago, Ill., OALR
*Fuller, T. E.(1915)
Texarkana Natl. Bk. Bldg., Texarkana, Tex., OALR
*Fulton, John F.(1920) 833 Lowry Bldg., St. Paul, Minn., OALR
Furgason, A. P.....Richards Blk., Lincoln, Neb., OALR
*Furstenberg, A. C.(1925)
330 S. State St., Ann Arbor, Mich., ALR
*Fust, John H. E.(1922).....154 W. 8th St., Erie, Pa., OALR

G

Gadbois, Arthur E.(1921).....Norfolk, Neb., OALR
*Gailey, Watson W.(1923).....Bloomington, Ill., OALR
Gale, M. Jean(1919).....Imperial Bldg., Denver, Colo., Op.
Gallagher, J. H. C.(1927)
317 Main St., Springfield, Mass., ALR
Gallaher, T. J.....California Bldg., Denver, Colo., ALR
*Galloway, T. C.(1927)....636 Church St., Evanston, Ill., OALR
Garber, J. M.....14 N. Mulberry St., Mansfield, O., OALR
Garraghan, Edw. F...25 E. Washington St., Chicago, Ill., OALR
Garrett, J. R.(1913).....Mt. Trust Bldg., Roanoke, Va., OALR
Gatewood, E. T.(1925) Professional Bldg., Richmond, Va., ALR
Gatewood, W. L....330 Park Ave.,New York City, N. Y., ALR
Gavin, Edward F.....1 S. Sheridan Rd., Waukegan, Ill., OALR
Geiger, C. W.(1904).....Cobb Bldg., Kankakee, Ill., OALR
George, Henry W.....19 N. Union St., Middletown, Pa., OALR
Gibson, R. D.(1907).....310 Wick Ave., Youngstown, O., OALR
Gifford, H.....Med. Arts Bldg., Omaha, Neb., Op.
*Gifford, S. R.(1919).....Med. Arts Bldg., Omaha, Neb., Op.
Gilbert, Frank Y.....148 Park St., Portland, Me., OALR
Gill, E. G(1919.....McBain Bldg., Roanoke, Va., ALR
*Gill, G. Bache(1923)
1624 Eye St., N. W., Washington, D. C., ALR
*Gill, George(1917)
Elyria Savings & Trust Bldg., Elyria, O., OALR

GILLETT, WILBUR G.....201 N. Main St., Wichita, Kas., Op.
*GILLETTE, D. F.(1925)....300 Union Blk., Syracuse, N. Y., Op.
GILLUM, J. R.(1920)...221 S. 6th St., Terre Haute, Ind., OALR
GIPNER, J. F.(1926)....53 Fitzhugh St., Rochester, N. Y., Op.
GITTINS, T. R.(1920).401 Davidson Bldg., Sioux City, Ia., OALR
GLASFORD, S.T.(1924).....Pekin, Ill., OALR
GLEASON, E. B.....2033 Chestnut St., Philadelphia, Pa., ALR
GLOGAU, O.....64 E. 91st St., New York City, N. Y., ALR
*GLOSSER, HERBERT H.(1912)
448 Franklin St., Buffalo, N. Y., Op.
GLOVER, S. P.....1118 Twelfth Ave., Altoona, Pa., OALR
GOAR, EVERETT LOGAN(1926)
1300 Walker Ave., Houston, Tex., Op.
GODDARD, H. M.....1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACK, L. J.(1911)
1012 N. Charles St., Baltimore, Md., Op.
*GOLDING, JOSEPH E.(1925)
170 Hancock St., Brooklyn, N. Y., Op.
GOLDMAN, H. G....57 W. 58th St., New York City, N. Y., ALR
*GOLDSMITH, P. G.....84 Carlton St., Toronto, Can., ALR
*GOLDSTEIN, M. A.(1896)
3858 Westminster Pl., St. Louis, Mo., ALR
GOODYEAR, H. M.(1924) 3029 Fairfield Ave., Cincinnati, O., ALR
GORDON, CHAS. H.(1926)...46 Deering St., Portland, Me., ALR
GORDON, J. S.(1919).141 Wisconsin St., Milwaukee, Wis., OALR
GORRELL, T. J. H..
16th & Vincennes Ave., Chicago Heights, Ill., OALR
GOSNEY, C. W.(1920).1009 Haas Bldg., Los Angeles, Cal., Op.
GOTTLIEB, J. M.814 West End Ave., New York City, N. Y., ALR
*GOURLEY, G. F.(1916).....Sinclair Bldg., Steubenville, O., OALR
GOUX, RAYMOND SALOT
545 David Whitney Bldg., Detroit, Mich., OALR
GRACEY, GEO. F.....209 State St., Harrisburg, Pa., OALR
*GRADLE, H. S.(1911)..58 E. Washington St., Chicago, Ill., Op.
GRAHAM, H. B.....490 Post St., San Francisco, Cal., ALR
GRANGER, W. B.(1920).....Emporia, Kan., OALR
*GRANT, FRED. E.
Kal. Natl. Bank Bldg., Kalamazoo, Mich., OALR
*GRANT, P. T.(1919)
1407 Grand Rapids Natl. Bk. Bldg., Grand Rapids, Mich., OALR
GRAY, E. B.(1927).....Mercer Bldg., New London, Conn., ALR
GREEN, A. S.....853 Hyde St., San Francisco, Cal., Op.
GREEN, JOHN(1905) 3720 Washington Ave., St. Louis, Mo., Op.
GREEN, L. D.....853 Hyde St., San Francisco, Cal., Op.
GREENE, JOSEPH BERRY(1923)
305 Haywood Bldg., Asheville, N. C., ALR
GREENFIELD, SAMUEL D.(1927)
169 New York Ave., Brooklyn, N. Y., ALR
*GREENWOOD, A.(1911)
82 Commonwealth Ave., Boston, Mass., Op.

GREEVER, B. L.(1920)
610 Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
GREGG, JOHN B...231 N. Summit Ave., Sioux Falls, S. D., OALR
GRIM, U. J.....30 N. Michigan Blvd., Chicago, Ill., ALR
GRISCOM, J. MILTON...2213 Walnut St., Philadelphia, Pa., Op.
GROSS, JULIUS E.(1920)
8030 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
GROSVENOR, L. N.(1907)
366 Dakota Ave., So. Huron, S. D., OALR
GROVE, W. E.(1914)
120 E. Wisconsin Ave., Milwaukee, Wis., OALR
GRUSHLOW, ISRAEL
271 Central Pk., W., New York City, N. Y., ALR
GSELL, J. F.(1920).....911 Beacon Bldg., Wichita, Kas., OALR
GUERINOT, A. J.(1918)
604 Westinghouse Bldg., Pittsburgh, Pa., OALR
GUGGENHEIM, L. K.....Carleton Bldg., St. Louis, Mo., ALR
GUNDELACH, C. A.,
825 University Club Bldg., St. Louis, Mo., ALR
GUTHRIE, A. L.(1910)
Amer. Natl. Bank Bldg., Oklahoma City, Okla., OALR
GUTTMAN, J....987 Madison Ave., New York City, N. Y., OALR
*GUYTON, B. S.(1918).....Oxford, Miss., OALR
GWINN, G. E.(1916)
704 Central Trust Bldg., San Antonio, Tex., OALR

H

*HAAS, FREDERICK J.(1920)
Masonic Bldg., Leavenworth, Kas., OALR
HACKNEY, F. J.(1913)
630 Volunteer Bldg., Chattanooga, Tenn., OALR
HADEN, HENRY C....Niels-Esperon Bldg., Houston, Tex., Op.
HAGEMAN, JOHN A.(1925) Highland Bldg., Pittsburgh, Pa., ALR
HAGER, W. A.....111 N. Lafayette St., South Bend, Ind., OALR
HAGLER, E. E.(1896).....Hagler Bldg., Springfield, Ill., OALR
*HALL, ALICE K.(1924).25 E. Washington St., Chicago, Ill., ALR
*HALL, G. C.(1907).....705 Brown Bldg., Louisville, Ky., OALR
HALLETT, G. DEWAYNE(1927)
274 W. 86th St., New York City, N. Y., Op.
HALSTED, FRED S....728 Metropolitan Bldg., Denver, Colo., OALR
*HAMLIN, FRED EUGENE(1926)
218 Shenandoah Life Bldg., Roanoke, Va., ALR
*HAMPTON, ROBT. R.,
804 Boston Bldg., Salt Lake City, Utah, ALR
HANCOCK, J. C.(1914)
135 Cambridge Pl., Brooklyn, N. Y., Op.
*HANDS, SIDNEY GILLOTT(1926)
602 1st Nat'l Bk. Bldg., Davenport, Ia., ALR
HANEY, WM. P....1500 Medical Arts Bldg., Omaha, Neb., OALR

HANS, C. L.(1921).....19 W. 7th St., Cincinnati, O., OALR
HANSEL, FRENCH KELLER(1926)
1045 Missouri Bldg., St. Louis, Mo., ALR
*HANSEN, ERLING W.,(1920)
910 Donaldson Bldg., Minneapolis, Minn., OALR
HARBERT, JOHN P.(1918)
135 N. Main St., Bellefontaine, O., OALR
HARDESTY, J. F.(1920)
1021 Missouri Bldg., St. Louis, Mo., Op.
HARDING, FREDERICK B.(1926)
25 S. 10th St., Allentown, Pa., OALR
HARDY, W. F.(1914).....Humboldt Bldg., St. Louis, Mo., Op.
HARGITT, CHARLES A.(1926)
17 Schermerhorn St., Brooklyn, N. Y., Op.
*HARKNESS, GORDON F.(1911)
Putnam Bldg., Davenport, Ia., OALR
*HARRELL, VOSS(1926).....Hofman Bldg., Detroit, Mich., ALR
HARRIS, C. M.(1910)
604 Johnstown Trust Bldg., Johnstown, Pa., OALR
HARRIS, H. B.....Fidelity Med. Bldg., Dayton, O., OALR
HARRIS, W. C.(1900).....7th and Race St., Cincinnati, O., ALR
HARRIS, W. ROBIN(1923)
Columbian Tower, Memphis, Tenn., ALR
HARRISON, W. G.....Empire Bldg., Birmingham, Ala., OALR
HARTLEY, F. A.....Fairbanks Bldg., Springfield, O., OALR
HARTSHORNE, ISAAC(1926)
30 W. 59th St., New York City, N. Y., Op.
HARTZEL, S. M.....Dollar Bank Bldg., Youngstown, O., OALR
HARVEY, N. D.....112 Waterman Ave., Providence, R. I., OALR
*HASELTINE, BURTON(1927)
122 S. Michigan Ave., Chicago, Ill., OALR
HASKELL, ALFRED W.....145 High St., Portland, Me., Op.
HASKIN, W. H.(1913)...40 E. 41st St., New York, N. Y., ALR
HASPEL, M. DAVID(1911)
603 Maison Blanche Bldg., New Orleans, La., OALR
HASTY, F. E.(1925)..420 Lambuth Bldg., Nashville, Tenn., OALR
HATCH, C. B.(1908).....3 W. Church St., Newark, O., OALR
*HAUER, A. M.(1916).....327 E. State St., Columbus, O., ALR
*HAUGHEY, WILFRID(1911)
303 Post Bldg., Battle Creek, Mich., OALR
HAWLEY, C. W.....Med. Arts Bldg., Chicago, Ill., OALR
HAWMAN, ERLE G...4051 N. Broad St., Philadelphia, Pa., OALR
HAYDEN, AUSTIN A.(1910)
25 E. Washington St., Chicago, Ill., OALR
*HAYS, HAROLD M.(1912)
22 W. 47th St., New York City, N. Y., ALR
HAZLETT, L. R.(1913)..Savings & Trust Bldg., Butler, Pa., OALR
HEALEY, JOHN F.(1924) 503 Delaware Ave., Buffalo, N. Y., ALR
HEARD, C. F.(1913).....813 Sassafrass St., Erie, Pa., OALR
HEARD, MARY K.....41 W. Main St., North East, Pa., Op.

*HEATH, PARKER (1924)
David Whitney Bldg., Detroit, Mich., Op.
HEBERT, ARTHUR W. . . . . Flood Bldg., San Francisco, Cal.
HECKEL, EDWARD B. (1902)
Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
HEEB, HARRY J. . . . . 204 Grand Ave., Milwaukee, Wis., OALR
HEED, C. R. . . . . 1205 Spruce St., Philadelphia, Pa., Op.
HEGGIE, N. M. (1905) . . . Buckman Bldg., Jacksonville, Fla., OALR
*HEITGER, J. D. (1905) . . . 808 Francis Bldg., Louisville, Ky., OALR
HEMPSTEAD, BERT E. . . . . Mayo Clinic, Rochester, Minn., ALR
HENNESSEY, WM. W. . . . . 333 Essex St., Salem, Mass., OALR
HENNING, CARL. . 1610 Eye St., N. W., Washington, D. C., Op.
HENNINGER, L. L. (1913)
205 Citizens Bk. Bldg., Pasadena, Cal., OALR
HENTON, G. E. (1918) . . . . . Morgan Bldg., Portland, Ore., OALR
HERRIMAN, L. L. (1913) . . . . . Alamosa, Colo., OALR
HESS, WM. L. (1904) . . . 1554 California St., Denver, Colo., OALR
HETRICK, L. E. (1923)
20 East 53rd St., New York City, N. Y., ALR
HETZEL, C. C. (1906) . . . . . Eccles Bldg., Ogden, Utah, OALR
HICKS, VONNIE MONROE (1926)
Professional Bldg., Raleigh, N. C., Op.
*HIGBEE, D. R. (1927) . . . . 3245 Fourth St., San Diego, Cal., ALR
HIGBEE, E. H. (1908)
417 Metropolitan Bldg., St. Louis, Mo., Op.
HIGGINS, R. P. (1924) . . . . . 20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. G. . . . . Wells Bldg., Milwaukee, Wis., OALR
HILL, C. E. (1919) . . . . . 160 Bloor St., W., Toronto, Can., Op.
*HILL, EMORY (1916) . . . Professional Bldg., Richmond, Va., Op.
HINMAN, E. E. (1924)
146 Washington Ave., Albany, N. Y., ALR
HITZ, H. B. . . . . Goldsmith Bldg., Milwaukee, Wis., ALR
HODSON, B. F. . . . . 418 Security Bldg., Miami, Fla., OALR
*HOFFMAN, JOHN N. (1927) . 308 Brant Bldg., Canton, Ohio, OALR
HOFFMAN, L. G. (1927) . 30 N. Michigan Ave., Chicago, Ill., OALR
HOFFMAN, WALTER F. (1920) . 817 Summit, Seattle, Wash., OALR
*HOFMAN, W. P. (1925) . . . . 707 Kahl Bldg., Davenport, Ia., OALR
HOGAN, W. L. (1924) . . . . . 750 Main St., Hartford, Conn., OALR
*HOGSHEAD, J. M. . . . 602 Georgia Ave., Chattanooga, Tenn., OALR
*HOGUE, D. W. (1908) . . . . . 34 W. High St., Springfield, O., OALR
HOHF, JULIUS (1915) . . . . . Clinic Bldg., Yankton, S. D., OALR
HOLDSWORTH, FRANK
206 Wilhelm Blk., Traverse City, Mich., OALR
HOLLOWAY, THOS. B. . 1819 Chestnut St., Philadelphia, Pa., Op.
HOLMES, BENJAMIN H. . . . . 435 Baker Blk., Racine, Wis., OALR
HOLT, E. E. JR. . . . . 723 Congress St., Portland, Me., Op.
*HOMPES, J. J. . . . . Security Mutual Bldg., Lincoln, Neb., OALR
*HOOD, T. C. (1904) Hume-Mansur Bldg., Indianapolis, Ind., Op.
HOPKINS, W. E. . . . 515 Park Ave., New York City, N. Y., OALR

*Hourn, George E.(1920)
511 University Club Bldg., St. Louis, Mo., ALR
Hovde, A. G.(1916). Board of Trade Bldg., Superior, Wis., OALR
*Howard, C. N.(1919)...210 S. Indiana St., Warsaw, Ind., OALR
Howard, E. F.(1919)
First Natl. Bank Bldg., Vicksburg, Miss., ALR
*Howard, Edward L.(1926)
First Natl. Bk., Ft. Worth, Tex., OALR
Howard, J. W.....Rialto Bldg., Kansas City, Mo., OALR
Howard, Lloyd G.....Council Bluffs, Iowa, OALR
Howard, W. H.(1923)
1009 Nicollet Ave., Minneapolis, Minn., OALR
Howe, A. W.(1922)
1616 Washington Bldg., Tacoma, Wash., OALR
Hubbard, A. E.....30 Redcliffe Rd., Buffalo, N. Y., Op.
*Hughes, J. A.(1927)
1220 Exchange Bldg., Memphis, Tenn., OALR
Hughes, L. W.(1925).....1019 Broad St., Newark, N. J., OALR
Hughes, T. E.(1922)..1000 W. Grace St., Richmond, Va., ALR
Hughes, W. F.,
401 Hume-Mansur Bldg., Indianapolis, Ind., Op.
Hurd, L. M.(1910) 39 E. 50th St., New York City, N. Y., ALR
Hurff, J. W.(1919)...86 Washington St., Newark, N. J., OALR
Hurley, E. D.(1914).....419 Boylston St., Boston, Mass., Op.
Hurst, V. Reeves(1923).....Longview, Tex., OALR
Husik, D. N.(1921)....1610 Spruce St., Philadelphia, Pa., ALR
Hussey, E. J.....245 Maple St., Holyoke, Mass., OALR

I

*Iglauer, S.....707 Race St., Cincinnati, O., ALR
Imperatori, C. J....17 E. 38th St., New York City, N. Y., ALR
Ingerman, S. M.(1909)
418 Central Park, W., New York City, N. Y., OALR
Ingersoll, J. M.(1908)....1020 Huron Rd., Cleveland, O., ALR
Inglis, Harry J.....43 Bay State Rd., Boston, Mass., ALR
Ingram, L. C.(1915).....Box 1711, Orlando, Fla., OALR
Irvin, Edgar H.....321 W. Rio Grande St., El Paso, Tex., OALR
Irvine, A. R.(1918)
709 Westlake Professional Bldg., Los Angeles, Cal., Op.
Irwin, Frank N.(1905)
114 E. 54th St., New York City, N. Y., Op.
Irwin, V. J.....1634 Main St., Springfield, Mass., OALR
Irwin, Vincent J., Jr.(1921)
1634 Main St., Springfield, Mass., OALR
Israel, Norma E.(1919)
2001 Niels Esperson Bldg., Houston, Tex., OALR
Israel, Sidney.2001 Niels Esperson Bldg., Houston, Tex., ALR
Ivins, H. M.(1910)
815 Merchants Natl. Bk. Bldg., Cedar Rapids, Ia., OALR

J

JACKLE, ARTHUR F.(1917)
63 E. Fourth St., Dunkirk, N. Y., Op.
JACKSON, CHEVALIER....128 S. 10th St., Philadelphia, Pa., L
*JACKSON, EDWARD(1902).....314–14th St., Denver, Colo., Op.
*JACKSON, J. D.(1920).....232 West 8th St., Erie, Pa., OALR
JACOBS, MAX W.(1916)....Missouri Bldg., St. Louis, Mo., Op.
JACOBS, S. M....1018 E. 163rd St., New York City, N. Y., OALR
JACOBY, D. P. A.(1912)....106 Touro St., Newport, R. I., Op.AL
JAMES, J. H.....Natl. City Bank Bldg., Mankato, Minn., OALR
JARECKY, HERMAN, 168 W. 86th St., New York City, N. Y., OALR
JEAN, GEO. W.....7 East Valerio, Santa Barbara, Cal., Op.
JENNINGS, J. ELLIS....807 Carleton Bldg., St. Louis, Mo., OALR
*JESSAMAN, L. W.(1914)
132 Union Ave., Framingham, Mass., OALR
JOBSON, G. B.(1903).....Franklin, Pa., OALR
*JOHNSON, A. E.(1919).....Watertown, S. D., OALR
JOHNSON, H. R.(1913)
512 Walnut Ave., Fairmont, W. Va., OALR
JOHNSON, OTIS H.....Morehead City, N. C., OALR
JOHNSON, THOMAS HOLLAND(1923)
30 W. 59th St., New York City, N. Y., Op.
*JOHNSTON, W. H.(1912).508 Laurel Bldg., Muscatine, Ia., OALR
*JOHNSTON, WILSON(1917) 611 Lovejoy St., Portland, Ore., OALR
JONES, C. C.(1924).....19 Garfield Pl., Cincinnati, O., ALR
*JONES, E. L.(1913)
First Natl. Bank Bldg., Cumberland, Md., OALR
*JONES, EDMUND LLOYD(1926)
Wheeling Clinic, Wheeling, W. Va., OALR
JONES, E. W.....44 Atlanta St., Lynn, Mass., Op.A
JONES, F. W.(1919).....Times Bldg., Alton, Ill., OALR
JONES, L. W.(1925)..53 S. Fitzhugh St., Rochester, N. Y., OALR
*JONES, U. G.(1927).....Johnson City, Tenn., ALR
*JONES, WM. S.(1927)
232 Sheridan Rd., Menominee, Mich., OALR
JORDAN, GEORGE T.(1921)
30 N. Michigan Blvd., Chicago, Ill., OALR
JOY, HAROLD HENRY(1926)
232 Brattle Rd., Syracuse, N. Y., Op.
*JOYNT, A. J.(1924).....Black Bldg., Waterloo, Iowa, OALR
JUDD, H. B.(1917)..47 E. 57th St., New York City, N. Y., ALR
JUDGE, HARRY V.(1920).....46 Willett St., Albany, N. Y., Op.

K

KAHN, M. E.(1925)...104 S. Michigan Ave., Chicago, Ill., ALR
KAUFFMAN, A. B.(1922)..Gurney Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. S.(1917) 1923 Spruce St., Philadelphia, Pa., ALR

*KEARBY, D. O. (1917)
709 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*KEATING, T. F. (1927)
5057 Woodward Ave., Detroit, Mich., OALR
KEELER, J. C. . . . . Med. Arts Bldg., Philadelphia, Pa., ALR
KEIM, W. F. (1924) . . . . . 25 Roseville Ave., Newark, N. J., ALR
KEITH, D. M. . . . . Wm. Brown Bldg., Rockford, Ill., ALR
KEITH, W. E. . . . . Waldheim Bldg., Kansas City, Mo., OALR
KELLER, S. A. . . . . Sioux Falls, S. D., OALR
KEMLER, JOS. (1922) . . . . . 1908 Eutaw Pl., Baltimore, Md., OALR
KENDALL, M. R. (1919) . . . . . Osborn Bldg., Cleveland, O., ALR
KENNEY, WM. L. . . . . Corby Forsee Bldg., St. Joseph, Mo., OALR
KENNON, W. G. (1924) . . Lambuth Bldg., Nashville, Tenn., OALR
KERRISON, P. D. . . . . 58 W. 56th St., New York City, N. Y., ALR
KETTELKAMP, FRED O. (1926)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
*KEY, BEN WITT (1914)
100 W. 59th St., New York City, N. Y., Op.
*KEY, SAM N. . . . . 320 Littlefield Bldg., Austin, Tex., OALR
*KEYS, MORTON J. (1921)
307 Jones Blk., Victoria, B. C., Canada, OALR
KIEFER, HUGO A. (1915)
Brockman Bldg., Los Angeles, Cal., OALR
*KIEHLE, F. A. . . . . 410 Med. Arts Bldg., Portland, Ore., OALR
*KILBOURNE, P. H. (1924) Fidelity Med. Bldg., Dayton, O., OALR
KIMBERLIN, J. W. (1915)
900 Rialto Bldg., Kansas City, Mo., Op.
KINCAID, J. H. . . . . 421 W. Church Ave., Knoxville, Tenn., OALR
*KING, CLARENCE (1910)
Union Central Bldg., Cincinnati, O., Op.
KING, CHAS. R. (1920) . . . . . 471 Spitzer Bldg., Toledo, O., OALR
KING, ED. . . . . 707 Race St., Cincinnati, O., ALR
KING, G. L. . . . . 537 E. Market St., Alliance, O., OALR
*KING, J. J. (1916) . . . . 40 E. 41st St., New York City, N. Y., ALR
KING, O. H. (1916) Dugan-Stuart Bldg., Hot Springs, Ark., OALR
*KIRBY, D. B. (1924) 30 W. 59th St., New York City, N. Y., Op.
KIRKENDALL, J. S. . . . . 315 N. Aurora St., Ithaca, N. Y., OALR
KIRKPATRICK, SAMUEL . . . . . Lauderdale Ave., Selma, Ala., OALR
KISER, J. D. . . . . Bartlesville, Okla., OALR
KISTLER, O. F. . . . . 43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
*KISTNER, F. B. . . . . 600 Mayer Bldg., Portland, Ore., ALR
KITTLESON, THEODORE (1915) . . . . . Fergus Falls, Minn., OALR
*KLINE, H. G. (1912) . . . . . University Blk., Syracuse, N. Y., ALR
KLINEDINST, JOHN FERD. (1906)
220 S. George St., York, Pa., Op.A
KLINGER, THOS. O. (1920)
318 Landers Bldg., Springfield, Mo., OALR
KNAPP, ARNOLD. . . . 10 E. 54th St., New York City, N. Y., Op.
KNAPP, B. . . . . Cleveland Life Bldg., Evansville, Ind., OALR
KNAPP, F. N. (1925) . . . . 329 Bradley Bldg., Duluth, Minn., OALR

*KNEEDLER, G. C.(1912) Professional Bldg., Pittsburgh, Pa., ALR
*KNODE, A. R.(1916)...708 Med. Arts Bldg., Omaha, Neb., ALR
KOLLOCK, C. W.....86 Wentworth St., Charleston, S. C., OALR
*KOOBS, H. J. G.(1920) 117½ W. Walnut St., Rogers, Ark., OALR
KOPETZKY, S. J....51 W. 73rd St., New York City, N. Y., ALR
KRAFT, OSCAR H...1524 N. Dearborn Pkwy., Chicago, Ill., Op.
KRAUSS, FREDERICK..1703 Chestnut St., Philadelphia, Pa., OALR
KREBS, ADOLPH(1914)....Jenkins Arcade, Pittsburgh, Pa., Op.
KRESS, GEO. H.....245 Bradbury Bldg., Los Angeles, Cal., OALR
*KRIEGER, G. M...701 Washington St., Michigan City, Ind., OALR
*KRIEGER, WM. A.(1919)
35 Market St., Poughkeepsie, N. Y., OALR
KRUG, ERNEST F.(1908)
12 W. 44th St., New York City, N. Y. Op.
KRUSKAL, I. D.(1925)..863 Prospect Pl., Brooklyn, N. Y., OP.
KULLY, B. M.....Med. Arts Bldg., Omaha, Neb., ALR

L

LAESSLE, HENRY A.(1922)
The Lenox, 13th & Spruce Sts., Philadelphia, Pa., ALR
LA FORCE, B. D.....1140 Beverley Rd., Altadena, Cal., OALR
LA FORCE, E. F.(1908)
Iowa State Bk. Bldg., Burlington, Ia., OALR
LAMB, F. W.(1908)
209 Provident Bk. Bldg., Cincinnati, O., OALR
LAMB, HARVEY(1920)
826 Metropolitan Bldg., St. Louis, Mo., Op.
LAMB, ROBT. SCOTT....Stoneleigh Ct., Washington, D. C., Op.
*LANCASTER, W. B.(1913)
520 Commonwealth Ave., Boston, Mass., Op.
LANDES, GEORGE A.....Parsons, Kas., OALR
LANDFRIED, C. J....607 Baronne Bldg., New Orleans, La., ALR
LANING, J. H.(1918)..907 Rialto Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M.(1902) Masonic Temple, Keokuk, Ia., OALR
LARGE, S. H.(1904)....2323 Prospect Ave., Cleveland, O., ALR
LARKIN, BERNARD J.(1916)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
LARSEN, CARL L.(1916)
1027 Lowry Bldg., St. Paul, Minn., OALR
LARUE, C. L.(1922).....Highland Clinic, Shreveport, La., OALR
LASALLE, ALBERT(1924)
3439 St. Denis St., Montreal, Can., OALR
LASSEN, FRITZ(1915).....Clinic Bldg., Pueblo, Colo., ALR
LA VIGNE, A. A.....471 Park Ave., New York City, N. Y., OALR
LAWRENCE, GRANVILLE A.,
Medical Arts Bldg., Philadelphia, Pa., Op.
LEA, JOS. A.....1125 Rialto Bldg., Kansas City, Mo., ALR
LEAVY, C. A.....Metropolitan Bldg., St. Louis, Mo., ALR

*LEBENSOHN, JAMES E.(1927)
3159 W. Roosevelt Rd., Chicago, Ill., OALR
LE COMPTE, E. D.(1927)
627 Boston Bldg., Salt Lake City, Utah., OALR
*LEDERER, FRANCIS L.(1925)
30 N. Michigan Blvd., Chicago, Ill., ALR
LEECH, J. W.....369 Broad St., Providence, R. I., OALR
LEFLER, ANNA B.(1918)
605 S. Broadway, Los Angeles, Cal., OALR
LEHAN, JAMES W.....1620–12th Ave., Greeley, Colo., OALR
LEJEUNE, F. E.(1927)
632 Maison Blanche Bldg., New Orleans, La., ALR
LEMERE, H. B.(1912)
330 Bankers Reserve Life, Omaha, Neb., OALR
LEMOINE, A. N.(1920) 1118 Rialto Bldg., Kansas City, Mo., Op.
LENKER, JOHN N.(1908)..1020 Huron Rd., Cleveland, O., ALR
LENT, E. J.....122 N. Lafayette Blvd., South Bend, Ind., OALR
*LERNER, MACY LEVI(1923)
332 Park Ave., Rochester, N. Y., Op.
LESTER, HARRY S.(1909)..420 Murray Bldg., Streator, Ill.
LEVENGOOD, H. W.(1927)
327 Wilshire Blvd., Santa Monica, Cal., OALR
*LEVY, LOUIS(1916)..1052 Madison Ave., Memphis, Tenn., ALR
LEVY, ROBERT(1896)...Metropolitan Bldg., Denver, Colo., ALR
LEWIS, A. C.(1916).....Exchange Bldg., Memphis, Tenn., OALR
LEWIS, EUGENE R.(1926)
730 S. Catalina, Los Angeles, Cal., ALR
LEWIS, FIELDING O.....259 S. 17th St., Philadelphia, Pa., ALR
*LEWIS, F. PARK.....454 Franklin St., Buffalo, N. Y., Op.
*LEWIS, GEORGE A.(1920).....Roundup, Mont., OALR
LEWIS, J. D.(1921)..1534 Estado St., Santa Barbara, Cal., OALR
LEWIS, J. P.(1915).....543 Boylston St., Boston, Mass., ALR
*LEWIS, WILLIAM W.(1921)
836 Lowry Bldg., St. Paul, Minn., OALR
*LEYDA, JAMES H.(1927)
406 Metropolitan Bldg., Denver, Colo., ALR
*LICHTENBERG, J. S.(1906) Rialto Bldg., Kansas City, Mo., Op.
LIDIKAY, C. J.(1920)..903 N. 7th St., Kansas City, Kans., OALR
LIFSCHUTZ, JACOB(1925) 133 S. Mason Ave., Chicago, Ill., OALR
LILLIE, HAROLD I.....Mayo Clinic, Rochester, Minn., ALR
LINN, E. G.(1913)...714 Equitable Bldg., Des Moines Ia., OALR
LINN, J. G.(1919)
7026 Jenkins Arcade Bldg., Pittsburgh, Pa., OALR
*LISCHKOFF, M. A.(1913)....Blount Bldg., Pensacola, Fla., OALR
*LITCHFIELD, J. T.(1921)
609 Yates Bldg., Minneapolis, Minn., OALR
LLOYD, A. W.(1915)....804 Lloyd Bldg., Hammond, Ind., OALR
*LLOYD, RALPH I.(1925)..14 Eighth Ave., Brooklyn, N. Y., Op.
*LOEB, CLARENCE(1907) 25 E. Washington St., Chicago, Ill., Op.

LOFTUS, JOHN EDWARD (1921)
605 Medical Arts Bldg., Philadelphia, Pa., ALR
LOKEY, H. M. . . . . Medical Arts Bldg., Atlanta, Ga., OALR
LOMBARDO, MELCHIORE (1923)
186 Meserole St., Brooklyn, N. Y., Op.
*LOOMIS, E. A. (1916) Donaldson Bldg., Minneapolis, Minn., OALR
*LOOPER, EDWARD ANDERSON (1924)
605 Park Ave., Baltimore, Md., OALR
*LORIE, ALVIN (1920) . . Commerce Bldg., Kansas City, Mo., ALR
LORING, J. B. . . . . 25 E. Washington St., Chicago, Ill., OALR
LOUCHERY, D. C. . . . . 212 W. Main St., Clarksburg, W. Va., OALR
LOVE, JOHN G. (1920) . . . . . Sedalia, Mo., OALR
LOWRY, B. W. (1925) 408 Citrus Exch. Bldg., Tampa, Fla., OALR
LUBMAN, MAX (1919)
17 E. 96th St., New York City, N. Y., ALR
LUEDDE, W. H. (1916) Metropolitan Bldg., St. Louis, Mo., Op.
LUKENS, CHARLES (1906) . . . . 316 Michigan St., Toledo, O., OALR
LUKENS, ROBT. McD.,
1308 Hunting Park Ave., Philadelphia, Pa., ALR
LUNDON, A. E. (1927)
119 Crescent St., Montreal, Canada, ALR
LUX, PAUL (1925) 409 Commerce Bldg., Kansas City, Mo., ALR
*LYLE, DONALD J. (1927) . . . . . 19 W. 7th St., Cincinnati, O., OALR
*LYMAN, HARRY W. (1923)
700 Carleton Bldg., St. Louis, Mo., ALR
*LYNCH, R. C. (1913)
Maison Blanche Bldg., New Orleans, La., ALR
LYONS, H. R. . . . . 30 N. Michigan Blvd., Chicago, Ill., ALR

M

MCALESTER, ANDREW W., JR.,
625 Bryant Bldg., Kansas City, Mo., Op.
*MCALLISTER, J. C. (1910) . . . 10 S. Mill St., Ridgeway, Pa., OALR
MCBEAN, GEO. M. . . . . 58 E. Washington St., Chicago, Ill., AL
*MCBRIDE, W. O. (1917)
2906 S. Fairfield Ave., Ft. Wayne, Ind., OALR
MCCANNELL, A. D. . . . . Colcord Bldg., Minot, N. D., OALR
MCCARTNEY, OLIVER P. (1920)
707 Elks Bldg., Kansas City, Kans., OALR
MCCARTY, VIRGIL W. . 800 Rialto Bldg., Kansas City, Mo.,
MCCASKEY, C. H.,
Continental Bank Bldg., Indianapolis, Ind., ALR
MCCAW, JOHN A. . . . . 418 Majestic Bldg., Denver, Colo., Op.
MCCLELLAN, W. E. (1927) . . . . . Trinidad, Colo., OALR
*MCCLELLAND, CARL C. (1923)
1857 David Whitney Bldg., Detroit, Mich., OALR
MCCONNELL, T. E.
Logan Trust Bldg., New Kensington, Pa., OALR

McCool, J. L.(1911)...909 Stevens Bldg., Portland, Ore., Op.
McCoy, J.....730 Fifth Ave., New York City, N. Y., ALR
McCready, J. H.....121 University Pl., Pittsburgh, Pa., ALR
*McCulloch, R. J.(1920)..102 College St., Toronto, Can., OALR
McDannald, C. E.(1927)
100 W. 59th St., New York City, N. Y., Op.
*McDermott, John J.(1924)
St. Joseph Sanitarium, St. Joseph, Mich., OALR
*McDiarmid, H. Oliver(1918)
35 Clement Block, Brandon, Manitoba, Can., OALR
McDonald, C. L.....964 Rose Bldg., Cleveland, O., OALR
McDonald, Dennis J.(1926)
17 E. 38th St., New York City, N. Y., ALR
McDowell, N. D.(1914)
275 Alexander St., Rochester, N. Y., OALR
McFarlane, Murray....190 Bloor St., E., Toronto, Can., OALR
McGinnis, Edwin...104 S. Michigan Blvd., Chicago, Ill., ALR
*McGivern, C. S.(1922)
101 S. Indiana Ave., Atlantic City, N. J., ALR
*McGuire, H. H....105 N. Braddock St., Winchester, Va., OALR
*McHenry, D. D.(1912)
604 Medical Arts Bldg., Oklahoma City, Okla., OALR
McKee, Claud W.(1917).Coulter Bldg., Greensburg, Pa., OALR
*McKee, Geo. J.(1922)
719 Westinghouse Bldg., Pittsburgh, Pa., ALR
*McKee, S. Hanford(1910)
158 Crescent St., Montreal, Can., Op.
McKelvey, A. D.....160 Bloor St., W., Toronto, Can., ALR
McKennan, Jas. W.
514 Washington Trust Bldg., Washington, Pa., Op.
McKeown, Elmer E...Metropolitan Bldg., Denver, Colo., OALR
*McKinney, A. R.(1922)
702 Second Natl. Bk. Bldg., Saginaw, Mich., OALR
McKinney, R.....1052 Madison Ave., Memphis, Tenn., ALR
McLaughlin, P. A.,
71 S. Washington St., Wilkes-Barre, Pa., OALR
McLaurin, John G.(1926)
1301 Medical Arts Bldg., Dallas, Texas, OALR
McLoone, John J.(1926) 611 Heard Bldg., Phoenix, Ariz., OALR
McMahon, Bernard John(1926)
604 Missouri Theatre Bldg., St. Louis, Mo., ALR
*McMurray, J. B.,
Washington Trust Bldg., Washington, Pa., OALR
*McNichols, W. A.(1925)
Dixon Nat'l. Bk. Bldg., Dixon, Ill., OALR
McReynolds, G. S.....State Bank Bldg., Temple, Tex., OALR
McReynolds, John O.,
907 Mercantile Bk. Bldg., Dallas, Tex., OALR
*McWilliams, Chas. A.(1927).....Gulfport, Miss., ALR
Macfarlane, P. B. 152 James St., S., Hamilton, Ont., Can., OALR

*MacKenzie, Alice V.(1919)
1724 Spruce St., Philadelphia, Pa., OALR
*MacKenzie, Geo. W.(1912)
1724 Spruce St., Philadelphia, Pa., OALR
MacLachlan, A. A.....May Bldg., Pittsburgh, Pa., ALR
*MacMillan, J. A.(1925)
1429 Stanley St., Montreal, Canada, Op.
Macnie, J. S.....74 S. 11th St., Minneapolis, Minn., OALR
Madden, Reed.....Allen Bldg., Xenia, O., OALR
Maggard, D. I.(1920)
1127 N. Emporia Ave., Wichita, Kan., OALR
Maghy, Chas. A.(1926)
1136 W. 6th St., Los Angeles, Cal., Op.
Maiden, S. D.(1918)
403 Bennett Bldg., Council Bluffs, Ia., OALR
*Maire, Lewis E.(1906)..710 Kresge Bldg., Detroit, Mich., OALR
Maloney, Thos. J....1032 Lowry Bldg., St. Paul, Minn., OALR
*Mann, R. H. T.(1913)
State Natl. Bk. Bldg., Texarkana, Tex., OALR
*March, Clara A.(1922)
465 Ashland Ave., Buffalo, N. Y., Op.
Markel, J. Clyde...Westinghouse Bldg., Pittsburgh, Pa., Op.
*Marlow, Searle B.(1924)
733 University Bldg., Syracuse, N. Y., Op.
Marquis, Geo. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
Marshall, George Guerin..Gryphon Bldg., Rutland, Vt., OALR
Marshall, Watson..121 University Pl., Pittsburgh, Pa., ALR
*Martin, Henry H.(1906)...247 Bull St., Savannah, Ga., OALR
Matheny, R. C. 405 Bk. of Galesburg Bldg., Galesburg, Ill., OALR
Matheson, J. P.(1915)
511 Independence Bldg., Charlotte, N. C., ALR
Matthews, Justus
1021 Metropolitan Bk. Bldg., Minneapolis, Minn., LR
Maudlin, L. O.
Chamber of Commerce Bldg., Greenville, S. C., OALR
Maumenee, A. E....710 Empire Bldg., Birmingham, Ala., OALR
Maxey, E. E.(1911)..609 Becker Bldg., Aberdeen, Wash., OALR
May, Chas. H.(1904)
698 Madison Ave., New York City, N. Y., Op.
*May, J. V.(1915).....1701 Main St., Marinette, Wis., OALR
May, James W.....802 Elks Bldg., Kansas City, Kan., Op.
Maybaum, J. L.(1916)
12 E. 86th St., New York City, N. Y., ALR
Meanor, Wm. C., 619 Westinghouse Bldg., Pittsburgh, Pa., Op.
Meierhof, E. L.(1912)
12 E. 86th St., New York City, N. Y., OALR
Mellinger, Wm. J.(1927)
120 E. Sola St., Santa Barbara, Cal., ALR
*Mercer, Ray(1920).....W. C. U. Bldg., Quincy, Ill., OALR
*Mercer, R. E.....David Whitney Bldg., Detroit, Mich., LR

MERRILL, H. G.(1917).....33 East 2nd St., Provo, Utah, OALR
MERRILL, W. HOWE(1906)
301 Essex St., Lawrence, Mass., OALR
MESSIER, A. E.(1925)..303 Park Bldg., Worcester, Mass., OALR
MESSINGER, H. C.....210 Angell St., Providence, R. I., Op.
METZ, ROY B.....1214 Guardian Bldg., Cleveland, O., Op.
*METZENBAUM, MYRON(1912)....Rose Bldg., Cleveland, O., ALR
MIDDLETON, A. B.....Pontiac, Ill., OALR
MIDDLETON, HARRY E.(1920).....Times Bldg., Alton, Ill., OALR
MILLER, ALICE R.(1919)
505 Guaranty Bldg., West Palm Beach, Fla., OALR
MILLER, C. E....305 Western Reserve Bldg., Muncie, Ind., OALR
MILLER, CLIFTON M.
Stuart Circle Hospital, Richmond, Va., OALR
MILLER, E. B.....2008 Walnut St., Philadelphia, Pa., Op.
MILLER, HUGH....520 Chambers Bldg., Kansas City, Mo., OALR
MILLETTE, J. W.....58 Cambridge Ave., Dayton, O., OALR
*MINER, S. G.(1896).....2641 E. Jefferson, Detroit, Mich., ALR
MINOR, C. L.....Fairbanks Bldg., Springfield, O., OALR
MINOR, H. F.....Randolph Bldg., Memphis, Tenn., OALR
*MINSKY, HENRY(1926)
215 W. 88th St., New York City, N. Y., Op.
MINTON, WM. H.....Bartlett Bldg., St. Joseph, Mo., OALR
MITCHELL, E. K...710 W. Lehigh Ave., Philadelphia, Pa., ALR
MITTENDORF, A. D..399 Park Ave., New York City, N. Y., Op.
MITTENDORF, W. KEITH,
115 E. 53rd St., New York City, N. Y., Op.
MITHOEFER, W.....19 Garfield Pl., Cincinnati, O., OALR
MOISE, ALLYN B.(1918)
407 Commercial Bldg., Shreveport, La., ALR
*MOLT, WM. F.(1911)
529 Bankers Trust Bldg., Indianapolis, Ind., ALR
MONAGHAN, D. G.....535 Majestic Bldg., Denver, Colo., Op.
MONAHAN, J. A.(1913)..181 Chestnut St., Clinton, Mass., OALR
*MONOSMITH, O. B.(1912)
Broadway & 8th St., Lorain, O., OALR
MONSON, S. H.....328 Anisfield Bldg., Cleveland, O., OALR
MOORE, GEO. H.(1919)....Schuylkill Bldg., Pottsville, Pa., OALR
MOORE, G. A.(1915).....Main St., Palmer, Mass., OALR
*MOORE, JOHN D.(1925)
6 Washington St., Bloomfield, N. J., OALR
*MOORE, T. K...416 Central Sav. & Trust Bldg., Akron, O., OALR
*MOORE, T. W.(1901) 1050 Fifth Ave., Huntington, W. Va., OALR
MOORE, WILLIAM FREDERICK
255 S. 16th St., Philadelphia, Pa., ALR
MORGAN, JAMES A.(1916)
49 Young Bldg., Honolulu, T. Hawaii, OALR
MORRIS, A. G.(1918)..303 Alexander St., Rochester, N. Y., ALR
MORRISON, FRANK A..224 N. Meridian, Indianapolis, Ind., Op.
MORRISON, WM. WALLACE(1926)
39 E. 50th St., New York City, N. Y., ALR

*MORSE, A. W.....507 Phoenix Bldg., Butte, Mont., OALR
*MORSMAN, L. W.(1920).205 Power Bldg., Hibbing, Minn., Op.
MORTON, HOWARD M.,
1111 Nicolett Ave., Minneapolis, Minn., Op.
*MORTON, J. B.(1924)..90 E. Warren Ave., Detroit, Mich., OALR
*MOSHER, H. P.....828 Beacon St., Boston, Mass., ALR
MOSS, HARVEY E.....1321 Rialto Bldg., Kansas City, Mo., OALR
MOTT, JOHN S.....214 Argyle Bldg., Kansas City, Mo., OALR
MOULTON, EVERETT C
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
MOULTON, H.,
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
*MULLIN, W. V.(1913)
Cleveland Clinic, Euclid Ave. & 93rd, Cleveland, O., ALR
MULSOW, J. E.(1921)
230 Wisconsin Ave., W., Milwaukee, Wis., OALR
MUNCASTER, S. B.1026 16th St., N. W., Washington, D. C., Op.
MUNCY, WM. M.(1914)..162 Angell St., Providence, R. I., OALR
*MUNDT, G. H.(1912)..25 E. Washington St., Chicago, Ill., OALR
MURDOCK, J. F.....Bersemer Bldg., Pittsburgh, Pa., Op.
MURPHY, J. G.....Murchison Bldg., Wilmington, N. C., OALR
MURPHY, W. E.....7th and Race Sts., Cincinnati, O., OALR
MURRAY, A. N.....4753 Broadway, Chicago, Ill., OALR
*MUSSUN, W. G.(1924)....610 Hanna Bldg., Cleveland, O., ALR
MYERS, ELMER A.,
407 Board of Trade Bldg., Superior, Wis.. OALR
MYERS, EUGENE LEE(1926)
207 Wall Bldg., St. Louis, Mo., ALR
*MYERS, JOHN L.(1920)
815 Shukert Bldg., Kansas City, Mo., OALR
MYERSON, M. C.(1923).198 Lincoln Place, Brooklyn, N. Y., ALR

N

NADEAU, E. G....Bellin-Buchanan Bldg., Green Bay, Wis., OALR
*NAFTZGER, J. B.....Davidson Bldg., Sioux City, Ia., OALR
*NANCE, WILLIS O.....30 N. Michigan Blvd., Chicago, Ill., OALR
NEEPER, EDW. R.,
Ex. Nat'l. Bank Bldg., Colorado Springs, Colo., Op.
NEHER, EDWIN MANSON(1926)
Boston Bldg., Salt Lake City, Utah, Op.
NEIGHBORS, CLARENCE A.,
R. F. D. No. 2, Box 87, Anaheim, Cal., OALR
NELSON, CHAS. F.....606 Schofield Bldg., Cleveland, O., OALR
NELSON, LOUIS A.....830 Lowry Bldg., St. Paul, Minn., Op.A
NELSON, LLOYD L.(1923).....Norfolk, Neb., OALR
NEW, GORDON B.....Mayo Clinic, Rochester, Minn., LR
NEWCOMB, JOHN R.,
410 Hume-Mansur Bldg., Indianapolis, Ind., Op.
NEWELL, WM. S..1209 Vermont Ave., Washington, D. C., Op.

*Newhart, Horace (1905)
Donaldson Bldg., Minneapolis, Minn., ALR
Newton, F. H. (1925) . . 907 Mercantile Bldg., Dallas, Tex., OALR
Newton, L. A. (1919)
709 Medical Arts Bldg., Oklahoma City, Okla., OALR
Nisselson, Max . . . . . 754 Beck St., New York City, N. Y., ALR
Noonan, W. J. (1905) . . . . . 4753 Broadway, Chicago, Ill., OALR
Norcross, E. R. (1924) . 30 N. Michigan Ave., Chicago, Ill., ALR
Northcott, Thomas A. (1926)
17 E. 38th St., New York City, N. Y., Op.
Norton, Roy R. . . . . Masonic Bldg., New Brighton, Pa., OALR
*Novak, Frank J., Jr. . . . 30 N. Michigan Blvd., Chicago, Ill., ALR
Noyes, G. L. . . . . Exchange Bank Bldg., Columbia, Mo., OALR
Nutter, C. F. . . . . 16 Amherst St., Nashua, N. H., Op.

O

Oberg, C. M. . . . . Syndicate Bldg., Minneapolis, Minn., OALR
*O'Brien, C. S. (1924) . . . . . Iowa City, Ia., Op.
*O'Brien, Steve A (1916)
1st Nat'l. Bank Bldg., Mason City, Ia., OALR
O'Connor, D.F. (1918) . . . . 671 Broad St., Newark, N. J., OALR
O'Connor, Roderic P. . . . 1904 Franklin St., Oakland, Cal., Op.
Oertel, T. E. . . . . 638 Green St., Augusta, Ga., OALR
Oesterling, H. E. (1922) . . . . . Box 454, Winter Park, Fla., OALR
Offutt, W. N. (1905)
406 Security Trust Bldg., Lexington, Ky., OALR
Olsho, S. L. (1912) . . . . . 235 S. 15th St., Philadelphia, Pa., OALR
Oppenheimer, Seymour
952 West End Ave., New York City, N. Y., OALR
Orcutt, D. C. (1912) . . 30 N. Michigan Ave., Chicago, Ill., Op.
O'Reilly, Charles A. (1911)
521 Med. Arts Bldg., Philadelphia, Pa., ALR
*O'Reilly, W. F. (1913) . . . 44 S. Common St., Lynn, Mass., OALR
O'Rourke, D. (1927) . . . 217 Imperial Bldg., Denver, Colo., Op.
*Orton, Henry B. . . . . 24 Commerce St., Newark, N. J., ALR
*O'Sullivan, T. J. (1927) . . . . . 10 Pine St., Portland, Me., ALR
*Overman, F. V. . . . Hume-Mansur Bldg., Indianapolis, Ind., ALR
Owen, F. S. . . . . Medical Arts Bldg., Omaha, Neb., OALR

P

Packard, Louis (1926) . . . . . Fort Collins, Colo., OALR
Page, Lafayette . . Hume-Mansur Bldg., Indianapolis, Ind., ALR
*Painter, A. M. (1918)
418 Bryant Bldg., Kansas City, Mo., ALR
Palmer, Arthur (1926)
2178 Broadway, New York City, N. Y., ALR
Palmer, Edw. M. (1919) 629 Beacon Bldg., Wichita, Kan., OALR
Park, J. Walter (1910) 32 N. Second St., Harrisburg, Pa., OALR

*PARKER, WALTER R..David Whitney Bldg., Detroit, Mich., Op.
PARRISH, MAJ. ROBERT E., M. C., U. S. A.,
125 Herbert Ave., San Antonio, Tex., ALR
PARSONS, J. G..313 Bank Electric Bldg., Lewistown, Mont., OALR
PATTERSON, ELLEN J.(1917)
121 University Pl., Pittsburgh, Pa., ALR
PATTERSON, JAS.A.(1904)
Burns Bldg., Colorado Springs, Colo., OALR
PATTERSON, W. E.....74 S. 11th St., Minneapolis, Minn., OALR
*PATTON, J. M.(1910)...Medical Arts Bldg., Omaha, Neb., Op.
PAYNE, A. L.(1908).Opera House Bldg., Eau Claire, Wis., OALR
PAYNE, R. J.(1925)
University Club Bldg., St. Louis, Mo., ALR
PEARLMAN, S. J.....29 E. Madison St., Chicago, Ill., ALR
*PEARSON, WM. W.(1908)
417 Bankers Trust Bldg., Des Moines, Ia., OALR
PEELER, C. N.(1920).....W. 7th St., Charlotte, N. C., OALR
*PEIRCE, HOWARD W.(1923)
704 Med. Arts Bldg., Detroit, Mich., ALR
PEMBER, AUBREY H.(1926)
500 W. Milwaukee St., Janesville, Wis. OALR
PENDEXTER, RALPH S.(1926)
1424 K St., N. W., Washington, D. C., Op.
PENTECOST, R. S.(1926)...184 Bloor St., Toronto, Canada, OALR
*PETER, L. C.(1914)
N. E. cor. Chestnut & 20th Sts., Philadelphia, Pa., Op.
PETERMAN, H. E.(1912)
512 Cathedral St., Baltimore, Md., OALR
PETERSON, R. A.(1920)
150 Fifth Ave., New York City, N. Y., OALR
PFAFFLIN, C. A,
Newton-Claypool Bldg., Indianapolis, Ind., OALR
PFINGST, A.....712 Brown Bldg., Louisville, Ky., Op.
PFINGSTEN, C. F.(1916)
6370 Waterman Ave., St. Louis, Mo., ALR
PHELPS, H. S.(1927)..322 S. Prospect St., Burlington, Vt., ALR
*PHELPS, KENNETH A.(1920)
730 La Salle Bldg., Minneapolis, Minn., OALR
PHILLIPS, W. C.....30 E. 58th St., New York City, ALR
PHILLIPS, WM. LINTON.131 Linwood Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PITKIN, C. E.....Osborn Bldg., Cleveland, O., ALR
*POLLOCK, H. L.(1911)....2551 N. Clark St., Chicago, Ill., ALR
PONTIUS, P.J.....1831 Chestnut St., Philadelphia, Pa., Op.
*POOS, E. E.(1927)..13300 Woodward Ave., Detroit, Mich., OALR
PORTER, E. H.(1912).....85 Madison St., Tiffin, O., OALR
*PORTER, JESSE B.(1920).....415 S. 2nd St., Elkhart, Ind., OALR
*PORTER, L. B.(1913)....199 Thayer St., Providence, R. I., OALR
*POST, LAWRENCE T.(1920)
520 Metropolitan Bldg., St. Louis, Mo., Op.

POST, M. HAYWARD (1918)
520 Metropolitan Bldg., St. Louis, Mo., Op.
POST, WINFRED B. (1921)
First National Bank Bldg., Carthage, Mo., OALR
*POTTER, GEO. B. (1921) .....471 Aquila Ct., Omaha, Neb., OALR
POTTER, L. F. .....68 Walnut St., New Bedford, Mass., OA
POTTER, PETER (1918) ...125 W. Granite St., Butte, Mont., OALR
POTTER, W. W. (1927) ...Arnstein Bldg., Knoxville, Tenn., OALR
POTTS, J. B. .....Medical Arts Bldg., Omaha, Neb., ALR
POWERS, EVERETT (1916)
Central Nat'l Bank Bldg., Carthage, Mo., OALR
*PRANGEN, AVERY DE H. (1919)
Mayo Clinic, Rochester, Minn., Op.
PRATT, FRED J. (1912)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
*PRATT, JOHN A. (1910)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
PRENDERGAST, D. A. .....850 Hanna Bldg., Cleveland, O., OALR
PRICE, NORMAN W. (1908)
445 3rd St., Niagara Falls, N. Y., OALR
*PROETZ, A. W. (1923) ....Beaumont Bldg., St. Louis, Mo., ALR
PROUD, WILLARD C. (1920)
Phys. & Surg. Bldg., St. Joseph, Mo., OALR
PROUT, A. W. (1920) .....370 E. Town St., Columbus, O., OALR
PYLE, WALLACE (1915) . 15 Exchange Pl., Jersey City, N. J., OA

Q

QUINBY, WM. O'G. (1919) ...14 James St., Newark, N. J., OALR
*QUINCY, J. E. (1927)
270 Commonwealth Ave., Boston, Mass., ALR
*QUITTNER, S. S. .....3912 Prospect Ave., Cleveland, O., OALR

R

RADCLIFFE, MCCLUNEY,
1906 Chestnut St., Philadelphia, Pa., Op.
RALLS, C. T. (1916) . . 1st Nat'l. Bank Bldg., Winfield, Kas., OALR
*RALSTON, WALLACE (1918)
1300 Walker Ave., Houston, Tex., Op.
RAMSEY, G. S. (1927) . 374 Mountain St., Montreal, Canada, Op.
RAVDIN, B. D. ....Citizens Natl. Bk. Bldg., Evansville, Ind., OALR
RAVDIN, M. .....Cititzens Natl. Bk. Bldg., Evansville, Ind., OALR
RAY, VICTOR (1901) .....19 W. 7th St., Cincinnati, O., Op.
REAVES, CHAS. R.,
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
*REAVES, W. P. (1913)
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
REBUCK, C. S. .....412 N. Third St., Harrisburg, Pa., OALR
RECTOR, A. E. (1915)
112 W. College Ave., Appleton, Wis., OALR

REEDER, J. E.(1916)...224 Davidson Bldg., Sioux City, Ia., OALR
REESE, MONROE D.(1926)..123 N. 8th St., Lebanon, Pa., OALR
*REESE, WARREN S.(1921) 230 S. 21st St., Philadelphia, Pa., Op.
*REGER, H. S.(1908)...Physicians Bldg., Jamestown, N. Y., OALR
REICHE, O. C.(1916)..Markle Bk. Bldg., Hazelton, Pa., OALR
REIM, HUGO.....417 Metropolitan Bldg., St. Louis, Mo., Op.
*REINEKE, GEO. F.(1926).....New Ulm, Minn., OALR
RENAUD, G. L....510 Joseph Campan Ave., Detroit, Mich., OALR
RENNER, W. S.(1900)..341 Linwood Ave., Buffalo, N. Y., ALR
*REPASS, R. E.(1920).....Ft. Lauderdale, Fla. ALR
REULING, F. H.(1924).....Black Bldg., Waterloo, Ia., OALR
*REYNOLDS, H. G.(1909)
City Natl. Bk. Bldg., Paducah, Ky., OALR
*REYNOLDS, J. S.(1918).La Salle Bldg., Minneapolis, Minn., OALR
RICE, CLARENCE PRENTICE.....Wahpeton, N. D., OALR
RICE, G. B.(1916) 270 Commonwealth Ave., Boston, Mass., ALR
*RICE, JOHN E.(1924).....390 Main St., Worcester, Mass., OALR
RICE, R. H.(1915).....331 Grove St., Milwaukee, Wis., OALR
*RICHARDS, L. G.(1924)
270 Commonwealth Ave., Boston, Mass., ALR
RICHARDSON, CHAS. W.,
1317 Connecticut Ave., Washington, D. C., ALR
RICHARDSON, S. A.(1925)
111 W. Adams St., Jacksonville, Fla., Op.
RICHIE, L. T.(1916)...McCormick Bldg., Trinidad, Colo., OALR
RIDEOUT, W. J.....701 State Bk. Bldg., Freeport, Ill., OALR
*RIDPATH, ROBT. F.,
N. E. cor. 18th & Chestnut Sts., Philadelphia, Pa., RL
*RIEGER, W. H.(1924).....Hanna Bldg., Cleveland, O., ALR
*RINDLAUB, ELIZABETH P.....620 Front St., Fargo, N. D., OALR
*RINDLAUB, JOHN H.....620 Front St., Fargo, N. D., OALR
*RINDLAUB, MARTIN P.....620 Front St., Fargo, N. D., OALR
RINGLE, CHAS. A.(1906)..Coronado Bldg., Greeley, Colo., ALR
RINGUEBERG, E. N. S.(1905)
13 Main St., Lockport, N. Y., Op.
RISDON, E. F.(1924)...30 Avenue Rd., Toronto, Canada, ALR
*ROBERTS, S. E.(1916)
1810 Federal Reserve Bank Bldg., Kansas City, Mo., ALR
ROBERTS, WM. H.....65 N. Madison Ave., Pasadena, Cal., Op.
*ROBERTSON, EDW. N.(1918)
132½ W. 6th St., Concordia, Kan., OALR
ROBERTSON, F. D.....Ashton Bldg., Grand Rapids, Mich., OALR
*ROBINSON, H. T.(1916)..Medical Bldg., Cumberland, Md., OALR
ROBINSON, R. E.(1920).....Waverly, Ia., OALR
*ROGERS, A. S.....105 S. Jefferson Ave., Saginaw, Mich., OALR
*ROGERS, J. T.....758 Sherbrooke St., W., Montreal, Can., ALR
*ROLLER, LOUIS A.(1920)
446 Paris Ave., S. E., Grand Rapids, Mich., Retired
ROMMEL, JOHN C....4601 N. Broad St., Philadelphia, Pa., Op.

*Rooker, A. M.(1920)
225 Gluck Bldg., Niagara Falls, N. Y., ALR
Roost, F. H.(1914).....Sioux City, Ia., OALR
Rosenbaum, J.....206 Bishop St., Montreal, Can., Op.
Roth, Albert W.(1920)
610 Security Natl. Bk. Bldg., Tulsa, Okla., OALR
*Rothschild, Harold J..239 Lowry Bldg., St. Paul, Minn., OALR
Rott, O. M.(1915).....Paulsen Bldg., Spokane, Wash., OALR
Row, Geo. S.(1911)
906 Hume-Mansur Bldg., Indianapolis, Ind., Op.
Rowland, John F.(1911)
Thompson Bldg., Hot Springs, Ark., OALR
Rowland, W. D.(1923).220 Clarendon St., Boston, Mass., Op.
Rubendall, C.(1915)..Medical Arts Bldg., Omaha, Neb., OALR
Ruby, F. M.....Hibbing, Minn., OALR
*Rucker, E. W.(1916)
926 Woodward Bldg., Birmingham, Ala., OALR
Rush, C. C....907 U. S. Natl. Bk. Bldg., Johnstown, Pa., OALR
Russell, E. L.(1920)
700 Argyle Bldg., Kansas City, Mo., OALR
Russell, J. A. Merle(1922).813 Sassafrass St., Erie, Pa., ALR
*Russell, R. E.(1924).....Waterloo, Iowa, OALR
Rust, E. G.....Hanna Bldg., Cleveland, O., Op.A
*Rutherford, C. W.(1925)
Pennway Bldg., Indianapolis, Ind., Op.
Ryan, L. M.(1927).....Wall Bldg., St. Louis, Mo., ALR
Ryder, D. R.....151 Rock St., Fall River, Mass., ALR
*Ryerson, F. L.(1925)....Fine Arts Bldg., Detroit, Mich., OALR

S

Sacco, Anthony(1920).....Union City, N. J., OALR
*Salinger, Samuel(1920)
25 E. Washington St., Chicago, Ill., ALR
Sallander, F. W.(1919)
204 Commerce Bldg., Sioux City, Ia., OALR
Sanderson, David D.(1916)..Funke Bldg., Lincoln, Neb., OALR
*Sanderson, H. H.(1906)
David Whitney Bldg., Detroit, Mich., OALR
Sartain, Paul J.(1921)
2006 Walnut St., Philadelphia, Pa., Op.
Satterlee, Richard H.
28 W. Chippewa St., Buffalo, N. Y., Op.
Sauer, W. E.(1904).....Humboldt Bldg., St. Louis, Mo., ALR
Sauer, W. W.(1910).....Box 416, Marietta, O., OALR
Savage, M. M.(1927)...1720 Eutaw Pl., Baltimore, Md., OALR
Scal, Joseph Coleman(1926)
44 W. 74th St., New York City, N. Y., ALR
Scales, H. L.(1920)
504-5 Rorabaugh-Wiley Bldg., Hutchinson, Kas., OALR

*SCALES, J. L.(1915)
Commercial Natl. Bk. Bldg., Shreveport, La., OALR
SCALES, J. W.(1896)...204½ Main St., Pine Bluff, Ark., OALR
SCARLETT, HUNTER W.(1919)
230 S. 21st St., Philadelphia, Pa., Op.
*SCHALL, L. A.(1927)
520 Commonwealth Ave., Boston, Mass., ALR
SCHAPPERT, N. L.(1916)
57 S. Washington St., Wilkes-Barre, Pa., OALR
SCHENCK, C. P.(1920)
604 Ft. Worth Natl. Bank Bldg., Fort Worth, Tex., OALR
SCHILD, E. H.(1906).....Brant Bldg., Canton, O., Op.
*SCHILLER, A. N.(1918)
249 West End Ave., New York City, N. Y., ALR
SCHLANSER, MAJOR A. E.(1921)
Army Medical Center, Washington, D. C., OALR
SCHLEGEL, HERMAN T.(1916)
501 Third St., Wausau, Wis., OALR
SCHLINDWEIN, G. WM.....138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN(1925)
1000 Park Ave., New York City, N. Y., Op.
SCHNEIDEMAN, T. B..2045 Chestnut St., Philadelphia, Pa., Op.
*SCHOLZ, R. P.....Metropolitan Bldg., St. Louis, Mo., ALR
SCHRANT, J. H.(1916)
Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
SCHULZE, E. C.(1926).1205 Med. Arts Bldg., Dallas, Tex., OALR
SCHURTER, M. A.(1924)
125 Temple Ave., Long Beach, Cal., OALR
SCHUSTER, FRANK P.(1927)
403 1st Natl. Bk. Bldg., El Paso, Tex., OALR
SCHUSTER, S. A.(1925).1st Natl. Bk. Bldg., El Paso, Tex., ALR
SCHUTZ, W. H.(1921)....Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, A. A.(1927)
203 W. 20th St., New York City, N. Y., ALR
SCHWARTZ, FREDERICK O.(1919)
701 Metropolitan Bldg., St. Louis, Mo., Op.
SCHWEINITZ, GEO. E. DE(1905)
1705 Walnut St., Philadelphia, Pa., Op.
SCHWENK, PETER N. K.
1417 N. Broad St., Philadelphia, Pa., Op.
SCOTT, J. R.(1920).....Ottawa, Kan., OALR
*SCOTT, MERLE E.(1926)..1st Natl. Bk. Bldg., Massillon, O., OALR
*SCROGGS, J. J.(1921).....218 E. Third St., Beaver, Pa., Op.A
SEAMAN, GILBERT E.,
407 Wells Bldg., Milwaukee, Wis., OALR
*SEARCY, H. B.(1912)
807 Greensboro Ave., Tuscaloosa, Ala., OALR
*SEARS, W. H.(1910).....514 Penn St., Huntingdon, Pa., OALR
SEAY, D. E.....907 Mercantile Bk. Bldg., Dallas, Tex., OALR
SECOY, F. L.(1922).....Frances Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W.(1917).....Ionia, Mich., OALR

SEDWICK, W. A. . . . . Imperial Bldg., Denver, Colo., Op.
SEEGMAN, SIMON (1918) . . Jenkins Arcade, Pittsburgh, Pa., ALR
SEELYE, W. K. (1920) . . 742 Stimson Bldg., Seattle, Wash., OALR
SELFRIDGE, G. L. . . . . 384 Post St., San Francisco, Cal., ALR
SELIGSTEIN, MILTON B. (1926)
801 Union Planters Bk. Bldg., Memphis, Tenn., OALR
SELLERS, L. M. (1920) . . Medical Arts Bldg., Dallas, Texas, ALR
SENER, WALTER J. . . . . Physicians Bldg., Philadelphia, Pa., Op.
SEWALL, E. C. (1910)
Stanford Univ. Hospital, San Francisco, Cal., ALR
*SEYDELL, E. M. (1916)
1021 First Natl. Bk. Bldg., Wichita, Kas., ALR
SHACKLETON, W. E. (1918)
2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J. (1913) . . . . . Starks Bldg., Louisville, Ky., OALR
SHAHAN, W. E. . . . 520 Metropolitan Bldg., St. Louis, Mo., Op.
SHANKLIN, E. M. . . . . 575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E. G. . . . . 135 S. 17th St., Philadelphia, Pa., Op.
SHAPERO, I. M. (1919) . . . . . 18 Gibbs St., Rochester, N. Y., OALR
SHARP, BENJ. S. (1926) . . 341 Thayer St., Providence, R. I., OALR
*SHARRETT, GEO. O. (1921) . 119 Bedford, Cumberland, Md., OALR
SHASTID, T. H. (1913)
10th Ave. Med. Bldg., Superior, Wis., Op.
SHAWHAN, GLENN E. (1913) . . . . . Nampa, Ida., OALR
*SHEA, J. J. (1919) . . . 1018 Madison Ave., Memphis, Tenn., OALR
*SHEAHAN, WM. L. (1926)
59 College St., New Haven, Conn., OALR
SHELLMAN, J. L. (1917) . . . . Lowry Bldg., St. Paul, Minn., OALR
*SHEMELEY, WM. G., JR., 1724 Spruce St., Philadelphia, Pa., OALR
SHERMAN, MAJ. A. ED., M.C., U. S. A. (1913)
Ft. Leavenworth, Kansas, OALR
*SHERMAN, ELBERT S. (1912) . 671 Broad St., Newark, N. J., Op.
SHIELDS, J. M. (1915) 264 Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE . . . . . Allen Bldg., Xenia, Ohio, ALR
SHIRAS, HOWARD H. (1920)
629 Guardian Bldg., Cleveland, O., Op.
*SHIREY, O. M. (1925) . . . . . Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND, U. S. Veterans' Hospital No. 67,
11th and Harrison Sts., Kansas City, Mo., OALR
SHOEMAKER, S. A. . . . . Bluffton, Ind.
SHOEMAKER, W. A. (1896)
1006 Carleton Bldg., St. Louis, Mo., Op.
SHORT, Z. N. . . . . Dugan-Stuart Bldg., Hot Springs, Ark., OALR
*SHREVE, OWEN M. (1905) . . . . . 162 W. 8th St., Erie, Pa., Op.
SHULTS, E. J. G. (1920) . . . . . Eldorado, Kansas, OALR
*SHUMAN, G. H. (1917) . . . . . Park Bldg., Pittsburgh, Pa., Op.
SHUMATE, D. L. . . . . 500 Argyle Bldg., Kansas City, Mo., OALR
*SHURLY, BURT R. . . . . 62 Adams Ave., W., Detroit, Mich., ALR
SHUTE, D. K. . . . . 1727 DeSales St., Washington, D. C., Op.
SIEGEL, FRANCIS X. (1922)
2600 Union Central Bldg., Cincinnati, O., OALR

SILBERNAGEL, C. E.(1918).283 E. State St., Columbus, O., OALR
*SIMONDS, O. F.....Hanna Bldg., Cleveland, O., Op.
*SIMPSON, H. LEE(1921)
1409 David Whitney Bldg., Detroit, Mich., ALR
*SIMPSON, W. L.(1911)
Phys. & Surg. Bldg., Memphis, Tenn., ALR
SINGLETON, E. M.(1897)
111 E. Church St., Marshalltown, Ia., OAL
*SKILLERN, R. H.(1910).1811 Spruce St., Philadelphia, Pa., R
*SKILLERN, S. R., JR.(1925)
2017 Spruce St., Philadelphia, Pa., OALR
SKIRBALL, JOSEPH J.(1927)
353 Commonwealth Ave., Boston, Mass.
*SLEIGHT, R. D.(1906).303 Post Bldg., Battle Creek, Mich., Op.
SLOAN, HENRY L.(1919)...6 W. 7th St., Charlotte, N. C., Op.
*SLOCUM, GEO.(1906)..311 S. State St., Ann Arbor, Mich., Op.
SLUDER, GREENFIELD
3542 Washington Ave., St. Louis, Mo., ALR
SMITH, ARTHUR E.,
1016 Donaldson Bldg., Minneapolis, Minn., OALR
SMITH, C. S.(1927)
214 N. Jefferson Ave., Springfield, Mo., OALR
SMITH, DORLAND(1910)
834 Myrtle Ave., Bridgeport, Conn., Op.A
SMITH, D. S.(1917)....State Bank Bldg., La Crosse, Wis., OALR
SMITH, E. TERRY.....36 Pearl St., Hartford, Conn., Op.
*SMITH, GEO. B.(1914).....Harlin Hospital, Rome, Ga., OALR
SMITH, H. A.(1925)
Colo. Bank & Trust Bldg., Delta, Colo., OALR
SMITH, J. M.....134½ N. Patterson St., Valdosta, Ga., OALR
SMITH, R. C.....Board of Trade Bldg., Superior, Wis., OALR
SMITH, S. McC.....1429 Spruce St., Philadelphia,Pa., ALR
SMITH, STANLEY.....121 University Pl., Pittsburgh, Pa., Op.
SMITH, VICTOR C.(1912)
722 Audubon Bldg., New Orleans, La., Op.
SNEED, CARL M.....506 W. Broadway, Columbia, Mo., OALR
*SNELL, ALBERT C.(1925)
53 S. Fitzhugh St., Rochester, N. Y., Op.
*SNYDER, W. H.....220 Michigan St., Toledo, O., OALR
SOMBERG, J. S.(1923)....60 E. 67th St., New York, N. Y., Op.
*SONNENSCHEIN, R.(1915).29 E. Madison St., Chicago, Ill., ALR
SPAKE, LAVERNE B.(1920)
320 Brotherhood Bldg., Kansas City, Kans., ALR
SPALDING, J. A.(1896)....627 Congress St., Portland, Me., Op.A
*SPENCER, FRANK R.(1904)
Physicians Bldg., Boulder, Colo., OALR
SPENCER, W. H., 1830 S. Rittenhouse Sq., Philadelphia, Pa., ALR
*SPENGLER, JOHN ARTHUR(1916)
423 S. Main St., Geneva, N. Y., Op.
*SPITZE, E. C.(1916)....Murphy Bldg., East St. Louis, Ill., Op.

*STAHLMAN, FRED C.....Waynesburg, Pa., OALR
STANBERRY, HENRY.....556 Doctors Bldg., Cincinnati, O., Op.
STANFORD, J. B.....1052 Madison Ave., Memphis, Tenn., Op.
STARK, JESSE B.(1925)
45 Park Ave., New York City, N. Y., Op.
STARK, WM. B.(1927).....Mayo Clinic, Rochester, Minn., ALR
STAUFFER, FRED(1912).133 1st Ave., Salt Lake City, Utah, OALR
*STEGMAN, LOUIE V.(1919) Sanitarium, Battle Creek, Mich., Op.
STEIM, J. M.(1914)
First Natl. Bk. Bldg., New Kensington, Pa., Op.
STEIN, O. J.....30 N. Michigan Blvd., Chicago, Ill., ALR
STEINFELD, A. L.(1922).....125–15th St., Toledo, O., OALR
STELLE, HARRY L...207 Commerce Bldg., Pittsburg, Kan., OALR
*STEVENSON, D. W.....165 E. Market St., Akron, O., OALR
*STEVENSON, WALTER(1915)..W. C. W. Bldg., Quincy, Ill., OALR
STEWART, WALTER E.(1920)
Tribune Bldg., Terre Haute, Ind., OALR
*STIEREN, E.(1905) 626 Union Trust Bldg., Pittsburgh, Pa., Op.
STILES, CHAS. M.(1908)
1130 Spruce St., Philadelphia, Pa., Op.
STILLSON, H.....Seaboard Bldg., Seattle, Wash., OALR
STIMSON, GEORGE W.(1919) Packard Apts., Warren, Ohio, OALR
*STITT, H. L.(1927).....656 Doctors Bldg., Cincinnati, O., ALR
STOCKLER, J. A.(1917)..332 North Fifth St., Reading, Pa., OALR
*STOKES, WM. H.(1926)...4105 Live Oak St., Dallas, Tex., Op.
*STOTTER, ARTHUR L.(1916)
1148 Euclid Ave., Cleveland, O., OALR
STOUT, PHILIP S....Medical Arts Bldg., Philadelphia, Pa., ALR
STOUTENBOROUGH, WM. A.(1927)
Medical Arts Bldg., Columbus, O., OALR
STRADER, GEO. L.(1904)
408 Hynds Bldg., Cheyenne, Wyo., OALR
STRAUSS, J. F.(1925)..104 S. Michigan Blvd., Chicago, Ill., ALR
STRICKLER, D. A.....Republic Bldg., Denver, Colo., Op.
STRONG, J. C.(1915)...810 Harrison Ave., Leadville, Colo., OALR
STROUD, EDWARD FRANK(1926)
805 Med. Arts Bldg., Dallas, Tex., ALR
STROUSE, FREDERICK M.(1920)
1301 Spruce St., Philadelphia, Pa., ALR
STROUT, E. S.(1907)...74 S. 11th St., Minneapolis, Minn., OALR
STROUT, G. E.(1921).Donaldson Bldg., Minneapolis, Minn., OALR
STUCKY, J. A.(1896) Guaranty Bank Bldg., Lexington, Ky., OALR
*STUEBER, F. G.(1905).....209 W. North St., Lima, O., OALR
*STUEBER, PAUL J.(1917).....209 W. North St., Lima, O., OALR
STURGES, LEIGH F.(1920)
121 E. 60th St., New York City, N. Y., ALR
STURM, S. A.(1916)
Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*SUKER, GEO. F.(1896).25 E. Washington St., Chicago, Ill., Op.
SULLIVAN, J. J., JR.(1923)
228 Scranton Life Bldg., Scranton, Pa., ALR

SULZER, G. A.(1907).....327 E. State St., Columbus, O., OALR
*SULZMAN, FRANK MALCOLM(1922)
1831 Fifth Ave., Troy, N. Y., OALR
SUMMERS, WM. S.(1927)
3752 Vicksburg, Detroit, Mich., OALR
SWAN, C. J.(1911).....636 Church St., Evanston, Ill., OALR
SWEET, ROBERT B.(1926)
Pacific S. W. Bk. Bldg., Long Beach, Calif., OALR
SWERDFEGER, E. B.(1927)
1008 Republic Bldg., Denver, Colo., ALR
SWETNAM, C. R. K.(1915) Masonic Temple, Prescott, Ariz., ALR

T

TANGEMAN, H. F.(1919)
606 Union Central Bldg., Cincinnati, O., ALR
*TAQUINO, GEORGE J.(1920)
208 Audubon Bldg., New Orleans, La., ALR
TARUN, WILLIAM.....605 Park Ave., Baltimore, Md., OALR
TAYLOR, CHARLES B.(1921).....Ottumwa, Ia., ALR
TAYLOR, LILLIAN E.(1905)
25 E. Washington St., Chicago, Ill., ALR
TAYLOR, LEWIS H.(1905)
83 S. Franklin St., Wilkes-Barre, Pa., OALR
*TAYLOR, M. C.(1915)....1206 May Bldg., Pittsburgh, Pa., Op.
THIGPEN, C. A.....401 So. Court St., Montgomery, Ala., OALR
THOMAS, C. D.(1914)
Central Natl. Bank Bldg., Peoria, Ill., OALR
*THOMAS, FRANCIS W.(1916)
327 E. State St., Columbus, O., OALR
THOMAS, H. C.(1926).....532 15th St., Oakland, Cal., OALR
THOMASON, H. E.....Rialto Bldg., Kansas City, Mo., ALR
THOMASSON, W.J.(1910)....942 York St., Newport, Ky., OALR
THOMPSON, E. H.....8 W. 9th St., Cincinnati, O., OALR
*THOMPSON, G. H.(1909)
18 Ashland St., North Adams, Mass., OALR
THOMPSON, HENRY M.(1912)
718 Bk. of Italy Bldg., 7th & Olive, Los Angeles, Cal., Op.
THOMPSON, J. J.(1907)...3 Park Ave., Mt. Vernon, N. Y., ALR
THOMPSON, PETER H.(1914)
308 Commonwealth Ave., Boston, Mass., Op.
TILDERQUIST, D. L.(1925)
425 Bradley Bldg., Duluth, Minn., OALR
TIMBERMAN, ANDREW.....318 E. State St., Columbus, O., OALR
TINDOLPH, L. W.....1st Natl. Bank Bldg., Olean, N. Y., ALR
TINGLEY, LOUISA PAINE(1913)
9 Massachusetts Ave., Boston, Mass., Op.
*TITSWORTH, GUY.....111 W. 4th St., Sedalia, Mo., OALR
*TIVNEN, RICHARD J.....30 N. Michigan Ave., Chicago, Ill., OALR
*TOBEY, GEO. L., JR. 270 Commonwealth Ave., Boston, Mass., ALR

TODD, GROVER CLEVELAND (1922)
307 Westinghouse Bldg., Pittsburgh, Pa., ALR
TOMASSENE, R. A. (1922)
Wheeling Bank & Trust Bldg., Wheeling, W. Va., OALR
*TOMLIN, W. S. (1912)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
TOMS, S. W. S. (1909) ....120 S. Broadway, Nyack, N. Y., OALR
TOOKER, C. W. (1907) .....Carleton Bldg., St. Louis, Mo., Op.
TOWNSEND, JOHN FERRARS (1923)
85 Wentworth, Cor. St. Phillips St., Charleston, S. C., OALR
*TRIBLE, G. B. (1920)
1801 Eye St., N. W., Washington, D. C., ALR
TRIMBLE, CLARENCE S. (1920)
405 Citizens Natl. Bank Bldg., Emporia, Kan., OALR
*TRIPP, IRA A. (1920) .....436 Rose Bldg., Cleveland, O., OALR
TRIPP, LEROY R. ....307 Security Bldg., Sioux City, Iowa, OALR
*TUCKER, GABRIEL (1923)
S. W. Cor. 34th & Spruce Sts., Philadelphia, Pa., B
*TUCKER, JAMES C. (1920) .....Beatrice, Neb., OALR
TUCKERMAN, W. C. (1911) 733 Osborn Bldg., Cleveland, O., Op.
TUCKERMAN, W. H. .....733 Osborn Bldg., Cleveland, O., OALR
TUREMAN, HERBERT G.,
1100 Rialto Bldg., Kansas City, Mo., ALR
TURNBULL, F. M. (1916) 25 N. 10th Ave., E. Duluth, Minn., ALR
*TURNER, H. H. (1914)
902 Professional Bldg., Pittsburgh, Pa., Op.
TURNER, OLIVER W. (1909) ..249 Water St., Augusta, Me., OALR

U

*UPHAM, HELEN F. (1907)
305 Third Ave., Asbury Park, N. J., OALR
UREN, C. T. (1919)
537 City Natl. Bank Bldg., Omaha, Neb., ALR
URNER, M. H. ....2600 Union Central Bldg., Cincinnati, O., OALR

V

VAIL, DERRICK T. .....24 E. 8th St., Cincinnati, O., Op.
VAIL, H. H. (1924) .....24 E. 8th St., Cincinnati, O., ALR
VAN KIRK, FRANK J., (1920)
Bell. Natl. Bank Bldg., Bellingham, Wash., OALR
VANKIRK, V. E. ....626 Union Trust Bldg., Pittsburgh, Pa., Op.
VAN METER, EUGENE R. (1927)
817 University Club Bldg., St. Louis, Mo., ALR
VAN POOLE, G. M. (1925)
Young Bldg., Honolulu, T. Hawaii, OALR
VAUGHAN, G. E. .....711 3rd St., Louisville, Ky., OALR
VAUGHN, E. H. .....Tyler, Tex., OALR

*VEASEY, C. A.(1905).....Paulsen Bldg., Spokane, Wash., OALR
VEASEY, C. A., JR.(1927)
404 Paulsen Bldg., Spokane, Wash.
VINSONHALER, F.(1902)
Urquhart Bldg., Little Rock, Ark., OALR
*VOIGT, C. B.(1912).....1702 Broadway, Mattoon, Ill., OALR
VON COLDITZ, G. THOMSEN
25 E. Washington St., Chicago, Ill., OALR
*VON DER HEYDT, ROBERT(1926)
25 E. Washington St., Chicago, Ill., Op.

W

*WAGENER, HENRY P.(1926) Mayo Clinic, Rochester, Minn., Op.
WAGERS, ARTHUR J.(1920)
The Lenox, 13th and Spruce Sts., Philadelphia, Pa., ALR
WAGNER, CARL B.(1912)..31 North State St., Chicago, Ill., OALR
WAGNER, W. A.(1927)
1003 Pere Marquette Bldg., New Orleans, La., ALR
*WALKER, CHAS. C.(1920)
604 Maccabee Bldg., Detroit, Mich., Op.
WALKER, C. E.....517 Jacobson Bldg., Denver, Colo., Op.
*WALKER, GEO. H.(1927)..Winona Clinic, Winona, Minn., OALR
WALKER, HERBERT S...30 N. Michigan Ave., Chicago, Ill., OALR
WALSH, JOSEPH MARK(1922).....Rapid City, S. D., OALR
WALTER, WILL.....1507 Hinman Ave., Evanston, Ill., OALR
WATKINS, EDWIN DIAL(1926)
1412 Exchange Bldg., Memphis, Tenn., OALR
WATSON, H. D.(1915) 151 Front St., Binghampton, N. Y., OALR
WATSON, JAMES A.,
500 Phys. & Surg. Bldg., Minneapolis, Minn.
*WATSON, R. S.,
120 N. Michigan Ave., Saginaw, W. S., Mich.
*WAUGH, J. M.,
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., OALR
*WEAVER, T. W.(1914).....201 Main St., Witchita, Kan., Op.
*WEED, HARRY M.(1908).196 Linwood Ave., Buffalo, N. Y., Op.
WEEKS, JOHN E.....Box 242, Oswego, Ore., Op.
*WEEKS, WEBB WILLIAM(1923)
20 E. 53rd St., New York City, N. Y., Op.
*WEIH, E. P.(1920).....Wilson Bldg., Clinton, Iowa, OALR
WEIL, A. I.....717 Audubon Bldg., New Orleans, La., ALR
WEIMER, EDGAR S.....332 Lehigh Ave., Pittsburgh, Pa., Op.
WEINSTEIN, M. A.(1927)
1309 Spruce St., Philadelphia, Pa., ALR
WEISSER, E. A.....806 May Bldg., Pittsburgh, Pa., Op.
*WELCH, ROBERT S. G.(1921)
46 State St. Circle, Annapolis, Md., OALR
WELLS, DAVID W.(1908)
Hotel Westminster, Boston, Mass., Op.

WELLS, GEO. S.....1525 State St., Santa Barbara, Cal., ALR
WELTY, C. F.(1926)....210 Post St., San Francisco, Cal., ALR
WENCKE, CARL G.,
Battle Creek Sanit., Battle Creek, Mich., ALR
*WENDEL, J. S.(1922)
744 David Whitney Bldg., Detroit, Mich., ALR
WESTLAKE, SAMUEL B....Humboldt Bldg., St. Louis, Mo., ALR
WETHERBEE, CHARLES(1926).....Twin Falls, Ida., OALR
WHEELER, ERNEST C.....Rust Bldg., Tacoma, Wash., OALR
*WHEELER, J. M.(1920)
30 W. 59th St., New York City, N. Y., Op.
WHEELER, MERRITT W.1027 Lowry Bldg., St. Paul, Minn., OALR
*WHELAN, G. L.(1925)..257 S. 16th St., Philadelphia, Pa., ALR
*WHERRY, W. P.(1910)
1500 Medical Arts Bldg., Omaha, Neb., OALR
WHISNANT, A. M.517 Professional Bldg., Charlotte, N. C., OALR
WHITAKER, J.(1917)....905 Congress Bldg., Miami, Fla., OALR
WHITE, E. H.(1912)
756 Sherbrooke St., W., Montreal, Can., ALR
WHITE, L. E.(1912)
390 Commonwealth Ave., Boston, Mass., ALR
WIENER, ALFRED(1909)
550 Park Ave., New York City, N. Y., OALR
WIENER, MEYER.....900 Carleton Bldg., St. Louis, Mo., Op.
*WILBUR, EDWARD P.,
403 Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
WILDER, WM. H.(1896)
122 S. Michigan Blvd., Chicago, Ill., Op.
WILLARD, L. M.(1915).....520 3rd St., Wausau, Wis., OALR
*WILLIAMS, HORACE J.(1922)
5908 Green St., Germantown, Philadelphia, Pa., ALR
*WILLIAMS, M. M.(1927)..148 Adams Ave., Scranton, Pa., Op.
WILLIAMS, W. C.(1906).....Jefferson Bldg., Peoria, Ill., OALR
*WILLIAMS, WALTON H.(1921)
Castell Bldg., Middletown, O., OALR
*WINGER, IRA B.(1927).....225 Michigan St., Toledo, O., OALR
*WINTER, GEORGE E.(1921)..44 Sun Bldg., Jackson, Mich., OALR
WINTER, JOHN A.(1923)
600 Fidelity Bldg., Duluth, Minn., OALR
*WISE, RALPH C.(1914).....Mansfield, O., OALR
WOLD, K. C.(1924)....1027 Lowry Bldg., St. Paul, Minn., OALR
WOLFE, CLAUDE T.(1916)..Francis Bldg., Louisville, Ky., OALR
*WOLFE, OTIS R.(1919)
415 Masonic Temple, Marshalltown, Iowa, OALR
WOOD, DOUGLAS....Donaldson Bldg., Minneapolis, Minn., OALR
WOODRUFF, FREDERICK E.,
Metropolitan Bldg., St. Louis, Mo., Op.
*WOODRUFF, H. W.....Morris Bldg., Joliet, Ill., OALR
*WOODRUFF, T. A.....Mercer Bldg., New London, Conn., Op.
WOODRUFF, WALTER STUART(1927)
421 Huguenot St., New Rochelle, N. Y., ALR

WOODS, HIRAM.....842 Park Ave., Baltimore, Md., Op.A
WOODS, R. H.....Neustadt Bldg., La Salle, Ill., Op.
WOODSON, J. M.(1914)
Cor. Avenue G and Fifth, Temple, Tex., OALR
*WOODWORTH, WM. P.(1927)
1424 Maccabees Bldg., Detroit, Mich., OALR
WORMLEY, H. R.....410 Brown Bldg., Rockford, Ill., OALR
WRIGHT, C. DARCY(1921)
307 Yates Bldg., Minneapolis, Minn., OALR
WRIGHT, J. B.....Professional Bldg., Raleigh, N. C., ALR
WRIGHT, J. W.....Central Natl. Bldg., Columbus, O., Op.
*WRIGHT, J. W. M.,
Hume-Mansur Bldg., Indianapolis, Ind., ALR
WRIGHT, WALTER WALKER(1926)
143 College St., Toronto, Canada, Op.
*WURDEMANN, HARRY V.....Cobb Bldg., Seattle, Wash., Op.
*WURTZ, W. J. M.(1913) 528 Delaware Ave., Buffalo, N. Y., ALR
WYLIE, C. B.....293 High St., Morgantown, W. Va., OALR

Y

YANKAUER, SIDNEY
121 E. 60th St., New York City, N. Y., ALR
YAZEL, HERMAN E.,
510-511 Grand Ave. Temple, Kansas City, Mo., OALR
YOUNG, CHARLES A.(1926)
Gill Mem. E., E. and Throat Hospital, Roanoke, Va., Op.
*YOUNG, H. B.(1897)..204 Jefferson St., Burlington, Iowa, OALR

Z

*ZEHNDER, A. CHARLES(1919)
188 Roseville Ave., Newark, N. J., Op.
ZEMER, S. G.....726 Security Mutual Bldg., Lincoln, Neb.
ZENTMAYER, WILLIAM..1506 Spruce St., Philadelphia, Pa., Op.
ZINKHAN, A. M.(1920)
The Rochambeau, Washington, D. C., ALR

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MAUMENEE, A. E. ..... 710 Empire Bldg., OALR
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SELMA

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BUCKLEY, J. H. ..... 1st National Bank Bldg., OALR
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MOULTON, EVERETT C. ..... Merchants National Bank Bldg., OALR
MOULTON, HERBERT ..... Merchants National Bank Bldg., OALR

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ROWLAND, JOHN F. ..... Thompson Bldg., OALR
SHORT, Z. N. ..... Dugan-Stuart Bldg., OALR

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Dayton, G. O.....245 Bradbury Bldg., OALR
Detling, Frank E.....907 Chapman Bldg., OALR
Dudley, W. H.....520 W. 7th St., OALR
Fisher, Carl.....538 W. 6th St., Op.A
Gosney, C. W.....1009 Haas Bldg., Op.
Irvine, A. R.....709 Westlake Professional Bldg., Op.
Kiefer, Hugo A.....Brockman Bldg., OALR
Kress, Geo. H.....245 Bradbury Bldg., OALR
Lefler, Anna B.....605 S. Broadway, OALR
Lewis, Eugene R.....730 S. Catalina, ALR
Maghy, Chas. A.....1136 W. 6th St., Op.
Thompson, Henry M.....718 Bk. of Italy Bldg., Op.

OAKLAND

O'Connor, Roderic P.....1904 Franklin St., Op.
Thomas, H. C.....532 15th St., OALR

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Roberts, Wm. H.....65 N. Madison Ave., Op.

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Higbee, D. R.....3245 Fourth St., ALR

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Barkan, Adolph.....Union Trust Bldg., OALR
Barkan, Hans.....490 Post St., Op.
Blake, Wm. F.....490 Post St., Op.
Brown, H. Alexander.....291 George St., Op.
Dolman, Percival.....490 Post St., Op.

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Graham, H. B.....490 Post St., ALR
Green, A. S.....853 Hyde St., Op.
Green, L. D.....853 Hyde St., Op.
Hebert, Arthur W.....Flood Bldg.
Selfridge, G. L.....384 Post St., ALR
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Welty, C. F.....210 Post St., ALR

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Wells, Geo. S.....1525 State St., ALR

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Fischer, V. B.....230 Mercantile Bank Bldg., OALR
Spencer, Frank R.....Physicians Bldg., OALR

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Patterson, Jas. A.....Burns Bldg., OALR

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Bane, Wm. M.....1005 Republic Bldg., OALR
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Black, Melville.....424 Metropolitan Bldg., Op.
Boyd, E. T.....823 Majestic Bldg., OALR
Brown, Morrow D.....432 Republic Bldg., ALR
Carmody, T. E.....Metropolitan Bldg., ALR
Collins, E. W.....508 Majestic Bldg., ALR
Cooper, C. E.....652 Metropolitan Bldg., ALR
Coover, D. H.....820 Metropolitan Bldg., Op.
Crisp, Wm. H.....Metropolitan Bldg., Op.
Finnoff, W. C.....217 Imperial Bldg., Op.A

FOLEY, J. W.....Majestic Bldg., OALR
FOSTER, J. McE.....Metropolitan Bldg., OALR
GALE, M. JEAN.....Imperial Bldg., Op.
GALLAHER, T. J.....California Bldg., ALR
HALSTED, FRED S.....728 Metropolitan Bldg., OALR
HESS, WM. L.....1554 California St., OALR
JACKSON, EDWARD.....314 14th St., Op.
LEVY, ROBERT.....Metropolitan Bldg., ALR
LEYDA, JAMES H.....406 Metropolitan Bldg., ALR
LOCHARD, L. B.....Metropolitan Bldg.
MCCAW, JOHN A.....418 Majestic Bldg., Op.
MCKEOWN, ELMER E.....Metropolitan Bldg., OALR
MONAGHAN, D. C.....535 Majestic Bldg., Op.
O'ROURKE, D.....217 Imperial Bldg., Op.
SEDWICK, W. A.....Imperial Bldg., Op.
SHIELDS, J. M.....264 Metropolitan Bldg., Op.
STRICKLER, D. A.....Republic Bldg., Op.
SWERDFEGER, E. B.....1008 Republic Bldg., ALR
WALKER, C. E.....517 Jacobson Bldg., Op.

FORT COLLINS

PACKARD, LOUIS..... OALR

GLENWOOD SPRINGS

COMEE, W. C..... OALR

GRAND JUNCTION

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LASSEN, FRITZ.....Clinic Bldg., ALR

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SHEAHAN, WM. L.....59 College St., OALR

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EAREL, A. M.....507 Willdon Bldg., OALR

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WOODRUFF, H. W.....Morris Bldg., OALR

KANKAKEE

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GLASFORD, S. T..... OALR

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FLOYD, T. W.....811 Peoria Life Bldg., OALR
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WILLIAMS, W. C.....Jefferson Bldg., OALR

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WORMLEY, H. R.....410 Brown Bldg., OALR

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PORTER, JESSE B.....415 S. 2nd St., OALR

EVANSVILLE

BROSE, L. D.....501 Upper First St., OALR
KNAPP, B.....Cleveland Life Bldg., OALR
RAVDIN, B. D.....Citizens Nat'l Bk. Bldg., OALR
RAVDIN, M.....Citizens Nat'l Bk. Bldg., OALR

FORT WAYNE

BARTHOLOMEW, A. C.....1020 Harrison St., OALR
BULSON, A. E., JR.....406 W. Berry St., OALR
MCBRIDE, W. O.....2906 S. Fairfield Ave., OALR

HAMMOND

LLOYD, A. W.....804 Lloyd Bldg., OALR
SHANKLIN, E. M.....575 Hohman St., OALR

INDIANAPOLIS

ADAMS, D. S.....508 Hume-Mansur Bldg., OALR
BARNHILL, J. F.....408 Pennway Bldg., ALR
BERRY, D. F.....Hume-Mansur Bldg., ALR
CARMACK, J. W.....505 Hume-Mansur Bldg., ALR
CHAPPELL, R. S.....305 Terminal Bldg., ALR
CLEVENGER, W. F.....Hume-Mansur Bldg., ALR
CRAFT, KENNETH L.....1003 Hume-Mansur Bldg., ALR

HOOD, T. C.....Hume-Mansur Bldg., Op.
HUGHES, W. F.....401 Hume-Mansur Bldg., Op.
KEARBY, D. O.....709 Hume-Mansur Bldg., ALR
LARKIN, BERNARD J.....Hume-Mansur Bldg., Op.
MCCASKEY, C. H.....Continental Bk. Bldg., ALR
MOLT, WM. F.....529 Bankers Trust Bldg., ALR
MORRISON, FRANK A.....224 N. Meridan, Op.
NEWCOMB, JOHN R.....410 Hume-Mansur Bldg., Op.
OVERMAN, F. V.....Hume-Mansur Bldg., ALR
PAGE, LAFAYETTE.....Hume-Mansur Bldg., ALR
PFAFFLIN, C. A.....Newton-Claypool Bldg., OALR
ROW, GEO. S.....906 Hume-Mansur Bldg., Op.
RUTHERFORD, C. W.....Pennway Bldg., Op.
TOMLIN, W. S.....Hume-Mansur Bldg., ALR
WRIGHT, J. W. M.....Hume-Mansur Bldg., ALR

MARION

BRAUNLIN, WM. H.....Marion Nat'l. Bank Bldg., OALR

MICHIGAN CITY

KREIGER, G. M.....701 Washington St., OALR

MUNCIE

MILLER, C. E.....305 Western Reserve Bldg., OALR

SOUTH BEND

BOONE, J. C.....213 Pythian Bldg., ALR
BOYD-SNEE, HARRY.....716 J. M. S. Bldg., ALR
EDWARDS, SLOCOMB R.....Christman Bldg., OALR
HAGER, W. A.....111 N. Lafayette St., OALR
LENT, E. J.....122 N. Lafayette Blvd., OALR

TERRE HAUTE

GILLUM, J. R.....221 S. 6th St., OALR
STEWART, WALTER E.....Tribune Bldg., OALR

WARSAW

HOWARD, C. N.....210 S. Indiana St., OALR

IOWA

BURLINGTON

LAFORCE, E. F.....Iowa State Bk. Bldg., OALR
YOUNG, H. B.....204 Jefferson St., OALR

CEDAR RAPIDS

BAILEY, FRED W.....Security Bldg., OALR
IVINS, H. M.....815 Merchants Natl. Bk. Bldg., OALR

CLINTON

WEIH, E. P.....Wilson Bldg., OALR

COUNCIL BLUFFS

DEAN, F. W.....Council Bluffs Clinic, OALR
HOWARD, LLOYD G.....OALR
MAIDEN, S. D.....403 Bennett Bldg., OALR

CRESTON

CHILDS, H. A.....Eagle Blk., OALR

DAVENPORT

HANDS, S. GILLOTT.....602–1st Natl. Bk. Bldg., ALR
HARKNESS, GORDON F.....Putnam Bldg., OALR
HOFMAN, W. P.....707 Kahl Bldg., OALR

DES MOINES

LINN, E. G.....714 Equitable Bldg., OALR
PEARSON, WM. W.....417 Bankers Trust Bldg., OALR

FORT DODGE

CHASE, S. B.....Carver Bldg., OALR

INDEPENDENCE

AGNEW, F. F.....120 Third Ave., S. E., OALR

IOWA CITY

BOILER, W. F.....OALR
DEAN, L. W.....12 S. Clinton St., OALR
O'BRIEN, C. S.....Op.

KEOKUK

LAPSLEY, ROBERT M.....Masonic Temple, OALR

MARSHALLTOWN

COBB, EDWIN.....Masonic Temple, OALR
FRENCH, R. F.....Masonic Temple, OALR
SINGLETON, E. M.....111 E. Church St., OAL
WOLFE, OTIS R.....415 Masonic Temple, OALR

MASON CITY

FALLOWS, HOWARD D.....Park Hospital Clinic, OALR
O'BRIEN, STEVE A.....First Natl. Bk. Bldg., OALR

MUSCATINE

JOHNSON, W. H.....508 Laurel Bldg., OALR

NEWTON

ENGLE, H. P.....200 Olive St., OALR

OTTUMWA

TAYLOR, CHARLES B.....OALR

SIOUX CITY

DECKER, J. C.....Francis Bldg., OALR
FRANCHERE, FRED'K. E.....4th and Nebraska St., OALR

GITTINS, T. R.....401 Davidson Bldg., OALR
NAFTZGER, J. B.....Davidson Bldg., OALR
REEDER, J. E.....224 Davidson Bldg., OALR
ROOST, F. H.....OALR
SALLANDER, F. W.....204 Commerce Bldg., OALR
SECOY, F. L.....Frances Bldg., OALR
TRIPP, LEROY R.....307 Security Bldg., OALR

WATERLOO

JOYNT, A. J.....Black Bldg., OALR
REULING, F. H.....Black Bldg., OALR
RUSSELL, R. E.....OALR

WAVERLY

ROBINSON, R. E.....OALR

KANSAS

ATCHISON

BRIBACH, EUGENE J.....212 Simpson Bldg., OALR

COFFEYVILLE

CHADWICK, IRA B.....4 Terminal Bldg., Op.

CONCORDIA

ROBERTSON, EDW. N.....132½ W. 6th St., OALR

EL DORADO

SHULTS, E. J. G.....OALR

EMPORIA

GRANGER, W. B.....OALR
TRIMBLE, CLARENCE S.....405 City Natl. Bk. Bldg., OALR

FORT LEAVENWORTH

SHERMAN, MAJ. A. ED., M.C., U. S. A.....OALR

HUTCHINSON

GREEVER, B. L.....610 Rorabaugh-Wiley Bldg., OALR
SCALES, H. L.....504 Rorabaugh-Wiley Bldg., OALR
SCHRANT, J. H.....Rorabaugh-Wiley Bldg., OALR

KANSAS CITY

LIDIKAY, C. J.....903 N. 7th St., OALR
McCARTNEY, OLIVER P.....707 Elks Bldg., OALR
MAY, JAMES W.....802 Elks Bldg., Op.
SPAKE, LAVERNE B.....320 Brotherhood, ALR

LEAVENWORTH

HAAS, FREDERICK J.....Masonic Bldg., OALR

OTTAWA

SCOTT, J. R.....OALR

PARSONS

LANDES, GEORGE A.....OALR

PITTSBURG

STELLE, HARRY L.....207 Commerce Bldg., OALR

TOPEKA

ALKIRE, H. L.....614 Kansas Ave., OALR
ALLEN, G. H.....Mills Bldg., OALR
ALLEN, GEO. V.....835 Kansas Ave., OALR

WICHITA

CHENEY, JAMES W.....702 Schweiter Bldg., OALR
DORSEY, J. G.....201 N. Main St., OALR
GILLETT, WILBUR G.....201 N. Main St., Op.
GSELL, J. F.....911 Beacon Bldg., OALR
MAGGARD, D. I.....1127 N. Emporia Ave., OALR
PALMER, EDW. M.....629 Beacon Bldg., OALR
SEYDELL, E. M.....1021 1st Natl. Bk. Bldg., ALR
WEAVER, T. W.....201 Main St., Op.

WINFIELD

RALLS, C. T.....First National Bank Bldg., OALR

KENTUCKY

BEREA

COWLEY, ROBERT HENRY.....OALR

COVINGTON

BLEDSOE, R. W.....1005 Madison Ave., OALR

FULTON

COHN, SELDON.....OALR

LEXINGTON

DEWEESE, CLARENCE.....154 N. Upper St., Op.
OFFUT, W. N.....406 Security Trust Bldg., OALR
STUCKY, J. A.....Guaranty Bk. Bldg., OALR

LOUISVILLE

BASS, A. L.....738 Francis Bldg., OALR
DABNEY, S. G.....Starks Bldg., OALR
DEAN, WALTER.....300 Frances Bldg., OALR
DULANEY, OCTAVUS.....811 Brown Bldg., ALR
HALL, G. C.....705 Brown Bldg., OALR
HEITGER, J. D.....808 Francis Bldg., OALR
PFINGST, A.....712 Brown Bldg., OALR
SHAFER, J. J.....Starks Bldg., OALR

VAUGHAN, G. E.....711 3rd St., OALR
WOLFE, CLAUDE T.....Francis Bldg., OALR

NEWPORT

THOMASSON, W. J.....942 York St., OALR

PADUCAH

REYNOLDS, H. G.....City National Bank Bldg., OALR

RICHMOND

DUNN, MURISON..... OALR

LOUISIANA

NEW ORLEANS

BAHN, CHAS. A.....1707 Pere Marquette Bldg., Op.
BLUM, HENRY.....1211 Maison Blanche Bldg., Op.
HASPEL, DAVID M.....603 Maison Blanche Bldg., ALR
LANDFRIED, C. J.....607 Baronne Bldg., ALR
LEJEUNE, F. E.....632 Maison Blanche Bldg., ALR
LYNCH, R. C.....Maison Blanche Bldg., ALR
SMITH, VICTOR C.....722 Audubon Bldg., Op.
TAQUINO, GEORGE J.....208 Audubon Bldg., Op.
WAGNER, W. A.....1003 Pere Marquette Bldg., ALR
WEIL, A. I.....717 Audubon Bldg., ALR

SHREVEPORT

LARUE, C. L.....Highland Clinic, OALR
MOISE, ALLYN B.....407 Commercial Bldg., ALR
SCALES, J. L.....Commercial Natl. Bk. Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W.....249 Water St., OALR

PORTLAND

BEACH, S. J.....704 Congress St., Op.
GILBERT, FRANK Y.....148 Park St., OALR
GORDON, CHAS. H.....46 Deering St., ALR
HASKELL, ALFRED W.....145 High St., Op.
HOLT, E. E. JR.....723 Congress St., Op.
O'SULLIVAN, T. J.....10 Pine St., ALR
SPALDING, J. A.....627 Congress St., Op.A

MARYLAND

ANNAPOLIS

WELCH, ROBERT S. G.....46 State St. Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., OALR
FRIEDENWALD, HARRY.....1212 Eutaw Pl., Op.A
FRIEDENWALD, J. S.....1212 Eutaw Pl., Op.
GOLDBACK, L. J.....1012 N. Charles St., Op.
KEMLER, JOS. J.....1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON.....605 Park Ave., OALR
PETERMAN, H. E.....512 Cathedral St., OALR
SAVAGE, M. M.....1720 Eutaw Pl., OALR
TARUN, WILLIAM.....605 Park Ave., OALR
WOODS, HIRAM.....842 Park Ave., Op.A

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
ROBINSON, H. T.....Medical Bldg., OALR
SHARRETT, GEO. O.....119 Bedford, OALR

MASSACHUSETTS

BOSTON

BRYANT, ALICE G.....502 Beacon St., ALR
CARVILLE, L. MAUD.....82 Commonwealth Ave., Op.
CHENERY, W. E.....377 Commonwealth Ave., ALR
CORBETT, J. J.....520 Beacon St., OALR
DERBY, GEO. S.....23 Bay State Road, Op.
FREEDMAN, L. M.....475 Commonwealth Ave., ALR
GREENWOOD, A.....82 Commonwealth Ave., Op.
HURLEY, E. D.....419 Boylston St., Op.
INGLIS, HARRY J.....43 Bay State Rd., ALR
LANCASTER, W. B.....529 Commonwealth Ave., Op.
LEWIS, J. P.....543 Boylston St., ALR
MOSHER, H. P.....828 Beacon St., ALR
QUINCY, J. E .....270 Commonwealth Ave., ALR
RICE, G. B.....270 Commonwealth Ave., ALR
RICHARDS, L. G.....270 Commonwealth Ave., ALR
ROWLAND, W. D.....220 Clarendon St., Op.
SCHALL, L. A.....520 Commonwealth Ave., ALR
SKIRBALL, J. J.....353 Commonwealth Ave., Op.
STANDISH, MYLES.....51 Hereford St., Back Bay, Op.
THOMPSON, PETER H.....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE.....9 Massachusetts Ave., Op.
TOBEY, GEO. L., JR.....270 Commonwealth Ave., ALR
WELLS, DAVID W.....Hotel Westminster, Op.
WHITE, L. E.....390 Commonwealth Ave., ALR

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

FRAMINGHAM

JESSAMAN, L. W.....132 Union Ave., OALR

GARDNER

BONE, HERMAN D.....19 Pleasant St., OALR

HOLYOKE

BURKE, JOHN R.....131 Chestnut St., OALR
HUSSEY, E. J.....245 Maple St., OALR

LAWRENCE

CONLON, F. A.....Bay State Bldg., OALR
MERRILL, W. HOWE.....301 Essex St., OALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....68 Walnut St., OA

NORTH ADAMS

THOMPSON, G. H.....18 Ashland St., OALR

NORTHAMPTON

COLLINS, J. D.....150 Main St., ALR
DOW, FRANK E.....4 West St., Op.

PALMER

MOORE, G. A.....OALR

SALEM

HENNESSEY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....67 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
GALLAGHER, J. H. C.....317 Main St., ALR
IRWIN, V. J.....1634 Main St., OALR
IRWIN, VINCENT J., JR.....1634 Main St., OALR

TAUNTON

BAKER, HARRY B.....59 Main St., OALR

WORCESTER

CAHILL, J. W.....390 Main St., OALR
CROSS, A. E.....390 Main St., Op.
ESTABROOK, CHAS. T.....857 Pleasant St., OALR
MESSIER, A. E.....303 Park Bldg., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ANN ARBOR

FURSTENBERG, A. C.....330 S. State St., ALR
SLOCUM, GEO.....311 S. State St., Op.

BATTLE CREEK

COLVER, B. N.....182 Manchester St., ALR
FRASER, R. H.....Battle Creek Sanitarium, ALR
HAUGHEY, WILFRID.....303 Post Bldg., OALR
SLEIGHT, R. D.....303 Post Bldg., Op.
STEGMAN, LOUIE V.....Sanitarium, Op.
WENCKE, CARL G.....Battle Creek Sanitarium, ALR

BAY CITY

BAKER, CHAS. H.....Crapo Block, OALR

DETROIT

AMBERG, EMIL.....622 Maccabee Bldg., ALR
BEATTIE, ROBERT.....David Whitney Bldg., Op.
BENTLEY, NEIL.....1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....10 Peterboro, OALR
BROWN, A. O.....742 Maccabee Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....David Whitney Bldg., OALR
CAMPBELL, M. D.....David Whitney Bldg., OALR
CLIFFORD, J. P.....1320 David Whitney Bldg., OALR
CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....1309 Stroh Bldg., ALR
FOWLER, WM.....1048 Maccabee Bldg., OALR
GOUX, RAYMOND SALOT.....545 David Whitney Bldg., OALR
HARRELL, VOSS.....Hofman Bldg., ALR
HEATH, PARKER.....David Whitney Bldg., Op.
KEATING, T. F.....5057 Woodward Ave., OALR
MCCLELLAND, CARL C.....1857 David Whitney Bldg., OALR
MAIRIE, LEWIS E.....710 Kresge Bldg., OALR
MERCER, R. E.....David Whitney Bldg., LR
MINER, S. G.....2641 E. Jefferson Ave., ALR
MORTON, J. B.....90 E. Warren Ave., OALR
PARKER, WALTER R.....David Whitney Bldg., Op.
PEIRCE, HORACE W.....704 Medical Arts Bldg., ALR
POOS, E. E.....13300 Woodward Ave., OALR
RENAUD, G. L.....510 Joseph Campan Ave., OALR
RYERSON, F. L.....Fine Arts Bldg., OALR
SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLY, BURT R.....62 Adams Ave., W., ALR
SIMPSON, H. LEE.....1409 David Whitney Bldg., ALR
SUMMERS, WM. S.....3752 Vicksburg, OALR
WALKER, CHAS. C.....604 Maccabee Bldg., Op.
WENDEL, J. S.....744 David Whitney Bldg., ALR
WOODWORTH, WM. P.....1424 Maccabee Bldg., OALR

ESCANABA

BOYCE, W. B.....1107 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED.....304 Gilbert Bldg., Op.
GRANT, P. T.....1407 Grand Rapids Natl. Bk. Bldg., OALR
ROBERTSON, F. D.....Ashton Bldg., OALR
ROLLER, LOUIS A.....446 Paris Ave., S. E., Retired

IONIA

SEDGWICK, O. W.....OALR

IRON MOUNTAIN

BOYCE, G. H.....408 E. D. St., OALR

JACKSON

WINTER, GEORGE E.....44 Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B.....308 Kalamazoo National Bank Bldg., OALR
GRANT, FRED E.....Kalamazoo National Bank Bldg., OALR
WILBUR, EDWARD P....403 Kalamazoo National Bank Bldg., OALR

LANSING

BURDICK, AUSTIN F.....416 Tussing Blk., OALR

MENOMINEE

ELWOOD, CALVIN R.....OALR
JONES, WM. S.....232 Sheridan Rd., OALR

MT. CLEMENS

CROMAN, JOS. M., JR.....OALR

ST. JOSEPH

MCDERMOTT, JOHN J.....St. Joseph Sanitarium, OALR

SAGINAW

MCKINNEY, A. R.....702–2nd Natl. Bk. Bldg., OALR
ROGERS, A. S.....105 S. Jefferson Ave., OALR
WATSON, R. S.....120 N. Michigan Ave.

TRAVERSE CITY

HOLDSWORTH, FRANK.....206 Wilhelm Blk., OALR

MINNESOTA

DULUTH

BRIGGS, F. W.....Fidelity Bldg., OALR
KNAPP, F. N.....329 Bradley Bldg., OALR
TILDERQUIST, D. L.....425 Bradley Bldg., OALR
TURNBULL, F. M.....25 N. 10th St., E., OALR
WINTER, JOHN A.....600 Fidelity Bldg., OALR

FERGUS FALLS

KITTLESON, THEODORE..... OALR

HIBBING

MORSMAN, L. W.....205 Power Bldg., OALR
RUBY, F. M..... OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BROWN, EDWARD J.....900 Nicollet Ave., OALR
CAMP, W. E.....716 Donaldson Bldg., OALR
CAMPBELL, ROBERT A.....520 La Salle Bldg., OALR
CLARK, H. S.....La Salle Bldg., OALR
DORGE, RICHARD IRVING.....76 Seymour Ave., S. E., OALR
FINK, WALTER H.....426 La Salle Bldg., OALR
HANSEN, ERLING W.....910 Donaldson Bldg., OALR
HOWARD, H.....1009 Nicollet Ave., OALR
LITCHFIELD, J. T.....609 Yates Bldg., OALR
LOOMIS, E. A.....Donaldson Bldg., OALR
MACNIE, J. S.....74 S. 11th St., OALR
MATTHEWS, JUSTUS.....1021 Metropolitan Bldg., LR
MORTON, HOWARD M.....1111 Nicollet Ave., Op.
NEWHART, HORACE.....Donaldson Bldg., ALR
OBERG, C. M.....Syndicate Bldg., OALR
PATTERSON, W. E.....74 S. 11th St., OALR
PHELPS, KENNETH A.....730 La Salle Bldg., OALR
PRATT, FRED J.....Metropolitan Bk. Bldg., OALR
PRATT, JOHN A.....Metropolitan Bk. Bldg., OALR
REYNOLDS, J. S.....La Salle Bldg., OALR
SMITH, ARTHUR E.....1016 Donaldson Bldg., OALR
STROUT, E. S.....74 S. 11th St., OALR
STROUT, G. E.....Donaldson Bldg., OALR
WATSON, JAMES A.....500 Phys. and Surg. Bldg.
WOOD, DOUGLAS.....Donaldson Bldg., OALR
WRIGHT, C. DARCY.....307 Yates Bldg., OALR

NEW ULM

REINEKE, GEO. F..... OALR

ROCHESTER

ANDERSON, CARL M.....Mayo Clinic, ALR
BENEDICT, WILLIAM L.....Mayo Clinic, Op.
FIGI, F. A.....Mayo Clinic, ALR
HEMPSTEAD, BERT E.....Mayo Clinic, ALR
LILLIE, HAROLD I.....Mayo Clinic, ALR
NEW, GORDON B.....Mayo Clinic, LR
PRANGEN, AVERY DE H.....Mayo Clinic, Op.
STARK, WM. B.....Mayo Clinic, ALR
WAGENER, HENRY P.....Mayo Clinic, Op.

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
BINGER, H. E.....824 Lowry Bldg., OALR
BOECKMANN, EGIL.....Lowry Bldg., OALR
BRAY, E. R.....342 Lowry Bldg., OALR
BROWN, JOHN C.....632 Lowry Bldg., OALR
BURCH, FRANK E.....754 Linwood Pl., Op.
CONNOR, CHAS. E.....Hamm Bldg., ALR
FOGARTY, CHAS. W.....1826 Summit Ave., OALR
FULTON, JOHN F.....833 Lowry Bldg., OALR
LARSEN, CARL L.....1027 Lowry Bldg., OALR
LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
MALONEY, THOMAS J.....1032 Lowry Bldg., OALR
NELSON, LOUIS A.....830 Lowry Bldg., Op.A
ROTHSCHILD, HAROLD J.....239 Lowry Bldg., OALR
SHELLMAN, J. L.....Lowry Bldg., OALR
WHEELER, MERRITT W.....1027 Lowry Bldg., OALR
WOLD, K. C.....1027 Lowry Bldg., OALR

WINONA

WALKER, GEO. H.....Winona Clinic, OALR

MISSISSIPPI

GULFPORT

MCWILLIAMS, CHAS. A.....ALR

OXFORD

GUYTON, B. S.....OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., Op.
EDWARDS, C. J.....First National Bank Bldg., OALR
HOWARD, E. F.....First National Bank Bldg., ALR

MISSOURI

CARTHAGE

POST, WINFRED B.....First National Bank Bldg., OALR
POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

NOYES, G. L.....Exchange Bk. Bldg., OALR
SNEED, CARL M.....506 W. Broadway, OALR

KANSAS CITY

ALLEN, CHAS. E.....712 Waldheim Bldg., OALR
ALTRINGER, A. N.....710 Rialto Bldg., ALR

BEAL, HOMER A.....1002 Argyle Bldg., ALR
BEIL, J. W.....Argyle Bldg., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Op.
BLAKESLEY, T. S.....Lathrop Bldg., OALR
CURDY, R. J.....1134 Rialto Bldg., Op.
CURRAN, E. J.....1805 Fed. Res. Bk. Bldg., Op.
DAVIS, HARRY B.....712 Bryant Bldg., OALR
FOSTER, HAL.....1014 Chambers Bldg., ALR
HOWARD, J. W.....Rialto Bldg., OALR
KEITH, W. E.....Waldheim Bldg., OALR
KIMBERLIN, J. W.....900 Rialto Bldg., Op.
LANING, J. H.....907 Rialto Bldg., ALR
LEA, JOS. A.....1125 Rialto Bldg., ALR
LEMOINE, A. N.....1118 Rialto Bldg., Op.
LICHTENBERG, J. S.....Rialto Bldg., Op.
LORIE, ALVIN.....Commerce Bldg., ALR
LUX, PAUL.....409 Commerce Bldg., ALR
MCALESTER, A. W., JR.....625 Bryant Bldg., Op.
MCCARTY, VIRGIL.....800 Rialto Bldg.
MILLER, HUGH.....520 Chambers Bldg., OALR
MOSS, HARVEY E.....1321 Rialto Bldg., OALR
MOTT, JOHN S.....214 Argyle Bldg., OALR
MYERS, JOHN L.....815 Shukert Bldg., OALR
PAINTER, A. M.....418 Bryant Bldg., ALR
ROBERTS, S. E.....1810 Federal Reserve Bank Bldg., ALR
RUSSELL, E. L.....700 Argyle Bldg., OALR
SCHUTZ, W. H.....Bryant Bldg., OALR
SHOEMAKER, FERDINAND,
U. S. Vet. Hosp., No. 67, 11th and Harrison Sts., OALR
SHUMATE, D. L.....500 Argyle Bldg., OALR
THOMASON, H. E.....Rialto Bldg., ALR
TUREMAN, HERBERT G.....1100 Rialto Bldg., OALR
YAZEL, HERMAN E.....510 Grand Ave. Temple, OALR

ST. JOSEPH

AMBROSE, ELMER C.....Logan Bldg., OALR
FORGRAVE, L. ROBERT.....204 Logan Bldg., Op.
KENNEY, WM. L.....Corby Forsee Bldg., OALR
MINTON, WM. H.....Bartlett Bldg., OALR
PROUD, WILLARD C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M.....537 Frisco Bldg., ALR
ARBUCKLE, MILLARD F.....1011 Beaumont Med. Bldg., ALR
BALL, JAMES MOORES.....4500 Olive St., Op.
BARCK, CARL.....Humboldt Bldg., Op.A
BARDENHEIER, F. G. A.....Frisco Bldg., Op.A
BLACK, W. D.....Metropolitan Bldg., ALR
BURNS, S. S.....Humboldt Bldg., ALR
CHARLES, JOS. W.....537 N. Grand Ave., Op.
COSTEN, J. B.....3720 Washington Ave., ALR

DONNELL, N. R.....Metropolitan Bldg., Op.
DYER, C. P.....233 Metropolitan Bldg., Op.
EWING, ARTHUR E.....Metropolitan Bldg., Op.
FREIMUTH, L. E.....234 University Club Bldg., ALR
GOLDSTEIN, M. A.....3858 Westminster Pl., ALR
GREEN, JOHN.....3720 Washington Ave., Op.
GUGGENHEIM, L. K.....Carleton Bldg., ALR
GUNDELACH, C. A.....825 University Club Bldg., ALR
HANSEL, FRENCH KELLER.....1045 Missouri Bldg., ALR
HARDESTY, J. F.....1021 Missouri Bldg., Op.
HARDY, W. F.....Humboldt Bldg., Op.
HIGBEE, E. H.....417 Metropolitan Bldg., Op.
HOURN, GEORGE E.....511 University Club Bldg., ALR
JACOBS, MAX W.....Missouri Bldg., Op.
JENNINGS, J. ELLIS.....807 Carleton Bldg., OALR
LAMB, HARVEY.....826 Metropolitan Bldg., Op.
LEAVY, C. A.....Metropolitan Bldg., ALR
LUEDDE, W. H.....Metropolitan Bldg., Op.
LYMAN, HARRY W.....700 Carleton Bldg., ALR
MCMAHON, BERNARD JOHN....604 Missouri Theatre Bldg., ALR
MYERS, EUGENE LEE.....Wall Bldg., ALR
PAYNE, R. J.....University Club Bldg., ALR
PFINGSTEN, C. F.....6370 Waterman Ave., ALR
POST, LAWRENCE T.....520 Metropolitan Bldg., Op.
POST, M. HAYWARD.....520 Metropolitan Bldg., Op.
PROETZ, A. W.....Beaumont Bldg., ALR
REIM, HUGO.....417 Metropolitan Bldg., Op.
RYAN, L. M.....Wall Bldg., ALR
SAUER, W. E.....Humboldt Bldg., ALR
SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O.....701 Metropolitan Bldg., Op.
SHAHAN, W. E.....520 Metropolitan Bldg., Op.
SHOEMAKER, W. A.....1006 Carleton Bldg., Op.
SLUDER, GREENFIELD.....3542 Washington Ave., ALR
TOOKER, C. W.....Carleton Bldg., Op.
VAN METER, EUGENE R.....817 University Club Bldg., ALR
WESTLAKE, SAMUEL B.....Humboldt Bldg., ALR
WIENER, MEYER.....900 Carleton Bldg., Op.
WOODRUFF, FREDERICK E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, JOHN G.....OALR
TITSWORTH, GUY.....111 W. 4th St., OALR

SPRINGFIELD

BAILEY, HAROLD.....840 Landers Bldg., OALR
COFFELT, THEODORE A.....508 Woodruff Bldg., OALR
KLINGER, THOS. O.....318 Landers Bldg., OALR
SMITH, C. S.....214 Jefferson Ave., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....Electric Light Bldg., OALR

BUTTE

DONOVAN, JNO. A.....507 Phoenix Bldg., OALR
MORSE, A. W.....507 Phoenix Bldg., OALR
POTTER, PETER.....125 W. Granite St., OALR

GREAT FALLS

COULTER, CHAS. F.....First Natl. Bank Bldg., OALR

LEWISTOWN

PARSONS, J. G.....313 Bank, Electric Bldg., OALR

ROUNDUP

LEWIS, GEORGE A..... OALR

NEBRASKA

BEATRICE

BAIRD, CHAS. G..... OALR
TUCKER, JAMES C..... OALR

COLUMBUS

CAMPBELL, C. H.....2904 15th St., OALR

KEARNEY

FOX, CHAS. H.....2119 Central Ave., OALR

LINCOLN

BLACK, PAUL.....514 1st Natl. Bk. Bldg., OALR
BROOKS, EARL B.....707 1st Natl. Bk. Bldg., OALR
CURTIS, W. L.....1503 S. 22nd St., OALR
DAYTON, W. L.....302 Funke Bldg., OALR
FURGASON, A. P.....Richards Block, OALR
HOMPES, J. J.....Security Mutual Bldg., OALR
SANDERSON, DAVID D.....Funke Bldg., OALR
ZEMER, S. G.....726 Security Mutual Bldg.

NORFOLK

GADBOIS, ARTHUR E..... OALR
NELSON, LLOYD L..... OALR

OMAHA

BANISTER, COL. J. M.....Bankers Reserve Life Bldg., OALR
BUSHMAN, L. B.....631 City Natl. Bk. Bldg., OALR
CALLFAS, W. F.....1620 Med. Arts Bldg., ALR
FAIRCHILD, NORA M.....Med. Arts Bldg., Op.
GIFFORD, HAROLD.....Med Arts Bldg., Op.
GIFFORD, S. R.....Med. Arts Bldg., Op.

HANEY, WM. P.....1500 Med. Arts Bldg., OALR
KNOVDE, A. R.....708 Med. Arts Bldg., ALR
KULLY, B. M.....Med. Arts Bldg., ALR
LEMERE, H. B.....330 Bankers Reserve Life Bldg., OALR
OWEN, F. S.....Med. Arts Bldg., OALR
PATTON, J. M.....Medical Arts Bldg., Op.
POTTER, GEO. B.....471 Aquila Ct., OALR
POTTS, J. B.....Med Arts Bldg., ALR
RUBENDALL, C.....Med. Arts Bldg., OALR
UREN, C. T.....537 City National Bank Bldg., ALR
WHERRY, W. P.....1500 Medical Arts Bldg., OALR

NEW HAMPSHIRE

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

FISHER, JAMES A.....Fitkin Bldg., OALR
UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. C.....124 S. Illinois Ave., ALR
MCGIVERN, C. S.....101 S. Indiana Ave., ALR

BLOOMFIELD

MOORE, JOHN D.....6 Washington St., OALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

JERSEY CITY

PYLE, WALLACE.....15 Exchange Pl., OA

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

BEGGS, W. F.....2 Lombardy St., OALR
CHATTIN, J. F.....671 Broad St., Op.
HUGHES, L. W.....1019 Broad St., OALR
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....25 Roseville Ave., ALR
O'CONNOR, D. F.....671 Broad St., OALR
ORTON, HENRY B.....24 Commerce St., ALR
QUINBY, W. O'G.....14 James St., OALR
SHERMAN, ELBERT S.....671 Broad St., Op.
ZEHNDER, A. CHARLES.....188 Roseville Ave., Op.

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

TRENTON

ADAMS, C. F.....34 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third Ave., OALR

UNION CITY

SACCO, A. G.....OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L.....1st Natl. Bk. Bldg., OALR

NEW YORK

ALBANY

BEDELL, ARTHUR J.....344 State St., Op.
DOWLING, J. IVIMEY.....116 Washington Ave., OALR
HINMAN, E. E.....146 Washington Ave., ALR
JUDGE, HARRY V.....46 Willett St., Op.

BINGHAMPTON

WATSON, H. D.....151 Front St., OALR

BROOKLYN

ARROWSMITH, HUBERT.....170 Clinton St., ALR
EVANS, J. N.....23 Schermerhorn St., Op.
GOLDING, JOSEPH E.....170 Hancock St., Op.
GREENFIELD, SAMUEL D.....169 New York Ave., ALR
HANCOCK, J. C.....135 Cambridge Pl., Op.
HARGITT, CHARLES A.....17 Schermerhorn St., Op.
KRUSKAL, I. D.....863 Prospect Pl., Op.
LLOYD, RALPH I.....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE.....186 Meserole St., Op.
MYERSON, M. C.....198 Lincoln Pl., ALR

BUFFALO

ANDREWS, H. D.....33 Allen St., Op.
BENNETT, ARTHUR G.....150 N. Pearl St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE.....438 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR
COTT, C. C.....483 Delaware Ave., ALR
COWPER, H. W.....543 Franklin St., Op.
EDSON, R.....498 Delaware Ave., Op.
FAIRBAIRN, JOHN.....503 Delaware Ave., ALR
FLAGG, J. D.....473 Virginia Ave., Op.A
GLOSSER, HERBERT H.....448 Franklin St., OALR

HEALY, JOHN F.....503 Delaware Ave., ALR
HOWE, LUCIEN.....520 Delaware Ave., Op.
HUBBARD, A. E.....30 Redcliffe Rd., Op.
LEWIS, F. PARK.....454 Franklin St., Op.
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON.....131 Linwood Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
SATTERLEE, RICHARD H.....28 W. Chippewa, Op.
WEED, HARRY M.....196 Linwood Ave., Op.
WURTZ, W. J. M.....528 Delaware Ave., ALR

CORTLAND

HIGGINS, R. P.....20 Court St., OALR

DUNKIRK

JACKLE, ARTHUR F.....63 E. 4th St., Op.

GENEVA

SPENGLER, JOHN ARTHUR.....423 S. Main St., Op.

GLENS FALL

FIELDING, F. G.....72 Ridge St., ALR

ITHACA

BULL, EDWARD L.....Saving Bk. Bldg., OALR
KIRKENDALL, J. S.....315 N. Aurora St., OALR

JAMESTOWN

REGER, H. S.....Physicians Bldg., OALR

LOCKPORT

RINGUEBERG, E. N. S.....13 Main St., Op.

MT. VERNON

THOMSON, J. J.....3 Park Ave., ALR

NEW ROCHELLE

BECK, A. L.....421 Huguenot St., OALR
WOODRUFF, WALTER STUART.....421 Huguenot St., ALR

NEW YORK CITY

ALBRIGHT, LT. COM. P. M., M.C., U. S. N.,
1st Brigade Marines, c/o Postmaster, OALR
ALMOUR, R. W.....51 W. 73rd St., ALR
AUERBACH, JULIUS.....120 W. 86th St., ALR
BALLIN, M. J.....108 E. 60th St., ALR
BEACH, B. S.....76 W. 86th St., Op.
BERENS, CONRAD.....30 E. 40th St., Op.
BRUDER, J.....229 W. 78th St., OALR
CARTER, W. W.....2 E. 54th St., ALR
COHEN, MARTIN.....1 W. 85th St., Op.
COHN, FELIX.....47 W. 87th St., ALR

COTT, D. N..... 141 St. Marks Pl., St. George, Staten Isl., ALR
DAVIS, A. EDWARD.....47 E. 57th St., Op.
DAVIS, G. E.....42 W. 77th St., OALR
DIXON, G. S.....218 Second Ave., R
DOHERTY, W. B.....150 W. 55th St., Op.
DUNNINGTON, J. H.....30 W. 59th St., Op.
ELLIS, Z. H.....30 W. 59th St., Op.
FREUDENTHAL, WOLFF.....24 W. 88th St., ALR
GATEWOOD, W. L.....330 Park Ave., ALR
GLOGAU, O.....64 E. 91st St., ALR
GOLDMAN, H. G.....57 W. 58th St., ALR
GOTTLIEB, J. M.....814 West End Ave., ALR
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
GUTTMAN, J.....987 Madison Ave., OALR
HALLETT, G. DEWAYNE.....274 W. 86th St., Op.
HARTSHORNE, ISAAC.....30 W. 59th St., Op.
HASKINS, W. H.....40 E. 41st St., ALR
HAYS, HAROLD M.....22 W. 47th St., ALR
HETRICK, L. E.....20 E. 53rd St., ALR
HOPKINS, W. E.....515 Park Ave., OALR
HURD, L. M.....39 E. 50th St., ALR
IMPERATORI, C. J.....17 E. 38th St., ALR
INGERMAN, S. M.....418 Central Park, W., OALR
IRWIN, FRANK N.....114 E. 54th St., Op.
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....168 W. 86th St., OALR
JOHNSON, THOMAS HOLLAND.....30 W. 59th St., Op.
JUDD, H. B.....47 E. 57th St., ALR
KERRISON, P. D.....58 W. 56th St., ALR
KEY, BEN WITT.....100 W. 59th St., Op.
KING, J. J.....40 E. 41st St., ALR
KIRBY, D. B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St., Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR
KRUG, ERNST F.....12 W. 44th St., Op.
LA VIGNE, A. A.....471 Park Ave., OALR
LUBMAN, MAX.....17 E. 96th St., ALR
MCCOY, JOHN.....730 Fifth Ave., ALR
MCDANNALD, C. E.....100 W. 59th St., Op.
MCDONALD, DENNIS J.....17 E. 38th St., ALR
MAY, CHAS. H.....698 Madison Ave., Op.
MAYBAUM, J. L.....12 E. 86th St., ALR
MAYER, EMIL.....666 West End Ave., ALR
MEIERHOF, E. L.....12 E. 86th St., OALR
MINSKY, HENRY.....215 W. 88th St., Op.
MITTENDORF, A. D.....339 Park Ave., Op.
MITTENDORF, W. KEITH.....115 E. 53rd St., Op.
MORRISON, WM. WALLACE.....39 E. 50th St., ALR
NISSELSON, MAX.....754 Beck St., ALR
NORTHCOTT, THOMAS A.....17 E. 38th St., Op.

OPPENHEIMER, SEYMOUR.....952 West End Ave., OALR
PALMER, ARTHUR.....2178 Broadway, ALR
PETERSON, R. A.....150 Fifth Ave., OALR
PHILLIPS, W. C.....30 E. 58th St., ALR
SCAL, JOSEPH COLEMAN.....44 W. 74th St., ALR
SCHILLER, A. N.....249 West End Ave., ALR
SCHLIVEK, KAUFMAN.....1000 Park Ave., Op.
SCHWARTZ, A. A.....203 W. 20th St., ALR
SOMBERG, J. S.....60 E. 67th St., Op.
STARK, JESSE B.....45 Park Ave., Op.
STURGES, LEIGH F.....121 E. 60th St., ALR
WEEKS, WEBB WILLIAM.....20 E. 53rd St., Op.
WHEELER, J. M.....30 W. 59th St., Op.
WIENER, ALFRED.....550 Park Ave., OALR
YANKAUER, SIDNEY.....121 E. 60th St., ALR

NIAGARA FALLS

PRICE, NORMAN W.....445 Third St., OALR
ROOKER, A. M.....225 Gluck Bldg., ALR

NYACK

TOMS, S. W. S.....120 S. Broadway, OALR

OLEAN

TINDOLPH, L. W.....First Natl. Bank Bldg., ALR

ONEIDA

CROCKETT, R. L.....302 Main St., Op.

POUGHKEEPSIE

KRIEGER, WM. A.....35 Market St., OALR

ROCHESTER

BARBER, F.....75 S. Fitzhugh St., Op.
CARROLL, GEO. G.....614 W. Main St., OALR
CLARK, L. H.....337 Monroe Ave., OALR
GIPNER, J. F.....53 Fitzhugh St., Op.
JONES, L. W.....53 S. Fitzhugh St., OALR
LERNER, MACY LEVI.....332 Park Ave., Op.
McDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....303 Alexander St., ALR
SHAPERO, I. M.....18 Gibbs St., OALR
SNELL, ALBERT C.....53 S. Fitzhugh St., Op.

SYRACUSE

BROWN, MORTIMER G.....802 University Bldg., ALR
BRITTEN, G. S.....University Blk., ALR
BRUST, H. O.....720 S. Crouse Ave., ALR
FOWLER, S. R.....University Bldg., ALR
GILLETTE, D. F.....300 University Blk., Op.
JOY, HAROLD HENRY.....232 Brattle Rd., Op.
KAUFFMAN, A. B.....Gurney Bldg., ALR

KLINE, H. G.....University Blk., ALR
MARLOW, SEARLE B.....733 University Bldg., Op.

TROY

SULZMAN, FRANK MALCOLM.....1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H.....252 Genesee St., OALR

WATERTOWN

ATKINSON, WALTER S.....168 Sterling St., Op.

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....101 Haywood St., OALR
GREENE, JOSEPH BERRY.....305 Haywood Bldg., ALR

CHARLOTTE

MATHESON, J. P.....511 Independence Bldg., ALR
PEELER, C. N.....W. 7th St., OALR
SLOAN, HENRY L.....6 W. 7th St., Op.
WHISNANT, A. M.....517 Professional Bldg., OALR

GREENSBORO

REAVES, CHAS. R...Reaves Infirmary, 117 W. Sycamore St., OALR
REAVES, W. P.....Reaves Infirmary, 117 W. Sycamore St., OALR

MOREHEAD CITY

JOHNSON, OTIS H..... OALR

NEW BERN

DANIELS, R. L..... OALR

RALEIGH

HICKS, VONNIE MONROE.....Professional Bldg., Op.
WRIGHT, J. B.....Professional Bldg., ALR

SALISBURY

BRAWLEY, R. V.....Op.

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W.....210 O'Hanlon Bldg., OALR

NORTH DAKOTA

FARGO

RINDLAUB, ELIZABETH P.....620 Front St., OALR
RINDLAUB, J. H.....620 Front St., OALR
RINDLAUB, M. P., JR.....620 Front St., OALR

MINOT

McCANNELL, A. D. ..... Colcord Bldg., OALR

WAHPETON

RICE, CLARENCE PRENTICE ..... OALR

OHIO

AKRON

BROWN, L. E. ..... 2nd Natl. Bank Bldg., OALR
MOORE, T. K. ..... 416 Central Sav. & Trust Bldg., OALR
STEVENSON, D. W. ..... 165 E. Market St., OALR

ALLIANCE

KING, G. I. ..... 537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H. ..... OALR
HARBERT, JOHN P. ..... 135 N. Main St., OALR

CANTON

HOFFMAN, JOHN N. ..... 308 Brant Bldg., OALR
SCHILD, E. H. ..... Brant Bldg., Op.

CINCINNATI

ABRAHAMSON, IRA ..... 605 Traction Bldg., Op.
ASBURY, MARY K. ..... 707 Race St., Op.
BEYER, A. G. ..... 19 Garfield Pl., OALR
BROWN, ALBERT LOUIS ..... 2600 Union Central Bldg., Op.
GOODYEAR, H. M. ..... 3029 Fairfield Ave., ALR
HANS, C. L. ..... 19 W. 7th St., OALR
HARRIS, W. C. ..... 7th & Race Sts., ALR
IGLAUER, SAMUEL ..... 707 Race St., ALR
JONES, C. C. ..... 19 Garfield Pl., ALR
KING, CLARENCE ..... Union Central Bldg., Op.
KING, ED. ..... 707 Race St., ALR
LAMB, F. W. ..... 209 Provident Bk. Bldg., OALR
LYLE, DONALD J. ..... 19 W. 7th St., OALR
MITHOEFER, W. ..... 19 Garfield Pl., OALR
MURPHY, W. E. ..... 7th and Race Sts., OALR
PHINNEY, F. D. ..... 22 W. 7th St., OALR
RAY, VICTOR ..... 19 W. 7th St., Op.
SIEGEL, FRANCIS X. ..... 2600 Union Central Bldg., OALR
STANBERRY, HENRY ..... 556 Doctors Bldg., Op.
STEWART, T. M. ..... 901 Union Trust Bldg., OALR
STITT, H. L. ..... 656 Doctors Bldg., ALR
TANGEMAN, H. F. ..... 606 Union Central Bldg., ALR
THOMPSON, E. H. ..... 8 W. 9th St., OALR
URNER, M. H. ..... 2600 Union Central Bldg., OALR
VAIL, DERRICK T. ..... 24 E. 8th St., Op.
VAIL, H. H. ..... 24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J.....8314 Euclid Ave., OALR
BRUNER, ABRAM BRENEMAN.....1214 Guardian Bldg.,Op.
BRUNER, WM. E.....Guardian Bldg., Op.
BURKE, T. A.....Rose Bldg., OALR
COGAN, J. E.....707 Rose Bldg., OALR
CUTLER, FRANKLIN E.....1026 Schofield Bldg., ALR
FORSYTHE, S. T.....235 Colonial Arcade, OALR
INGERSOLL, J. M.....Osborn Bldg., ALR
KENDALL, M. R.....Osborn Bldg., ALR
LARGE, S. H.....2323 Prospect Ave., ALR
LENKER, JOHN N.....1020 Huron Rd., ALR
MCDONALD, C. L.....942 Rose Bldg., OALR
METZ, ROY B.....1214 Guardian Bldg., Op.
METZENBAUM, MYRON.....Rose Bldg., ALR
MONSON, S. H.....328 Anisfield Bldg., OALR
MULLIN, W. V..Cleveland Clinic, Euclid Ave., and 63rd St., ALR
MUSSUN, W. G.....610 Hanna Bldg., ALR
NELSON, CHAS. F.....606 Schofield Bldg., OALR
PITKIN, C. E.....Osborn Bldg., ALR
PRENDERGAST, D. A.....850 Hanna Bldg., OALR
QUITTNER, S. S.....3912 Prospect Ave., OALR
RIEGER, W. H.....Hanna Bldg., ALR
RUST, E. G.....Hanna Bldg., Op.A
SHACKLETON, W. E.....2323 Prospect Ave., Op.
SHIRAS, HOWARD H.....629 Guardian Bldg., Op.
SHIREY, O. M.....Osborn Bldg., ALR
SIMONDS, O. F.....Hanna Bldg., Op.
STOTTER, ARTHUR L.....1148 Euclid Ave., OALR
TRIPP, IRA A.....436 Rose Bldg., OALR
TUCKERMAN, W. C.....733 Osborn Bldg., Op.
TUCKERMAN, W. H.....733 Osborn Bldg., OALR
WAUGH, J. M....Cleveland Clinic, Euclid Ave. at 93rd St., OALR

COLUMBUS

BEATTY, H. G.....681 E. Broad St., OALR
BROWN, JOHN E.....370 E. Town St., OALR
CLARK, I. G.....186 E. State St., OALR
HAUER, A. M.....327 E. State St., ALR
PROUT, A. W.....370 E. Town St., OALR
SILBERNAGEL, C. E.....283 E. State St., OALR
STOUTENBOROUGH, WM. A.....Medical Arts Bldg., OALR
SULZER, G. A.....327 E. State St., OALR
THOMAS, FRANCIS W.....327 E. State St., OALR
TIMBERMAN, ANDREW.....318 E. State St., OALR
WRIGHT, J. W.....Central National Bldg., Op.

DAYTON

BINKLEY, ROY S.....530 Fidelity Bldg., OALR
BLACKBURN, W. J.....661 Reibold Bldg., OALR
DUTROW, H. V.....1040 Fidelity Bldg., OALR

FARMER, A. G.....Fidelity Medical Bldg., OALR
FOUTS, JOHN D.....910 Fidelity Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, P. H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....58 Cambridge Ave., OALR

ELYRIA

GILL, GEORGE.....Elyria Sav. & Trust Bldg., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts.

LIMA

STUEBER, F. G.....209 W. North St., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....Broadway and 8th St., OALR

MANSFIELD

GARBER, J. M.....14 N. Mulberry St., OALR
WISE, RALPH C..... OALR

MARIETTA

SAUER, W. W.....Box 416, OALR

MARION

BRICKLEY, DANIEL W.....196 W. Center St., OALR

MASSILLON

SCOTT, MERLE E.....1st Natl. Bk. Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H.....Castell Bldg., OALR

NEWARK

HATCH, C. B .....3 W. Church St., OALR

SANDUSKY

BLISS, C. B.....411 Columbus Ave., OALR

SPRINGFIELD

EASTON, J. C.....Fairbanks Bldg., OALR
HARTLEY, F. A.....Fairbanks Bldg., OALR
HOGUE, D. W.....34 W. High St., OALR
MINOR, C. L.....Fairbanks Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDERDYCE, WM.....513 Madison Ave., OALR
ALTER, F. W.....Toledo Medical Bldg., OALR
DENMAN, IRA O.....425 Ohio Bldg., OALR
KING, CHAS. R.....471 Spitzer Bldg., OALR
LUKENS, CHARLES.....316 Michigan Ave., OALR
SNYDER, W. H.....220 Michigan St., OALR
STEINFELD, A. L.....125–15th St., OALR
WINGER, IRA B.....225 Michigan St., OALR

WARREN

STIMSON, GEORGE W.....Packard Apt., OALR

XENIA

MADDEN, REED.....Allen Bldg., OALR
SHIELDS, LAWRENCE.....Allen Bldg., ALR

YOUNGSTOWN

BEARD, H. J.....510 Dollar Bank Bldg., OALR
GIBSON, R. D.....310 Wick Ave., OALR
HARTZELL, S. M.....Dollar Bank Bldg., OALR

ZANESVILLE

CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D.....OALR

GUTHRIE

BARKER, CHAS. B.....OALR

McALESTER

DAVIS, J. E.....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M.....404 Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E.....503 Medical Arts Bldg., OALR
FERGUSON, E. S.....902 Medical Arts Bldg., OALR
GUTHRIE, A. L.....Amer. Natl. Bank Bldg., OALR
McHENRY, D. D.....604 Medical Arts Bldg., OALR
NEWTON, L. A.....709 Medical Arts Bldg., OALR

TULSA

BRASWELL, JAMES C.....708 Mayo Bldg., OALR
COOK, W. ALBERT.....506 Palace Bldg., OALR
ROTH, ALBERT W.....610 Security Natl. Bk. Bldg., OALR

OREGON

LA GRANDE

BOUVY, L. B.....OALR

OSWEGO

WEEKS, JOHN E.....Op.

PORTLAND

AINSLIE, GEORGE.....606 Oregonian Bldg., OALR
BOUVY, HARRY.....800 Pittock Blk., OALR
BRUERE, G. E.....Journal Bldg., OALR
DAVIS, RALPH F.....1104 Selling Bldg., OALR
FENTON, RALPH A.....806 Medical Arts Bldg., OALR
HENTON, G. E.....Morgan Bldg., OALR
JOHNSTON, WILSON.....611 Lovejoy St., OALR
KIEHLE, F. A.....410 Medical Arts Bldg., OALR
KISTNER, F. B.....600 Mayer Bldg., ALR
MCCOOL, J. L.....909 Stevens Bldg., Op.

PENNSYLVANIA

ALLENTOWN

HARDING, FREDERICK B.....25 S. 10th St., OALR

ALTOONA

GLOVER, S. P.....1118–12th Ave., OALR

BEAVER

SCROGGS, J. J.....218 E. 3rd St., Op.

BETHLEHEM

CHASE, W. D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....357 N. Main St., OALR
HAZLETT, L. R.....Savings & Trust Bldg., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.

ERIE

DENNIS, DAVID N.....221 W. 9th St., Op.
DENNIS, EDWARD P.....309 W. 10th St., OALR
FUST, J. H. E.....154 W. 8th St., OALR
HEARD, C. F.....813 Sassafrass St., OALR

JACKSON, J. D.....232 W. 8th St., OALR
RUSSELL, J. A. MERLE.....813 Sassafrass St., ALR
SCHLINDWEIN, G. WM.....138 W. 9th St., OALR
SHREVE, OWEN M.....162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B..... OALR

GREENSBURG

FAIRING, J. W.....205 Tribune Bldg., OALR
McKEE, CLAUD W.....Coulter Bldg., OALR

HARRISBURG

FARNSLER, H. HERSHEY.....1438 Market St., OALR
GRACEY, GEO. F.....209 State St., OALR
PARK, J. WALTER.....32 N. 2d St., OALR
REBUCK, C. S.....412 N. 3d St., OALR

HAZELTON

REICHE, O. C.....Markle Bk. Bldg., OALR

HUNTINGDON

SEARS, W. H.....514 Penn St., OALR

JOHNSTOWN

BARKER, O. G. A.....804 Johnstown Trust Bldg., Op.
HARRIS, C. M.....604 Johnstown Trust Bldg., OALR
RUSH, C. C.....907 U. S. Natl. Bk. Bldg., OALR

LEBANON

REESE, MONROE D.....123 N. 8th St., OALR

MIDDLETOWN

GEORGE, HENRY W.....19 N. Union St., OALR

NEW BRIGHTON

NORTON, ROY R.....Masonic Bldg., OALR

NEW KENSINGTON

McCONNELL, T. E.....Logan Trust Bldg., OALR
STEIM, J. M.....1st Natl. Bk. Bldg., Op.

NORTH EAST

HEARD, MARY K.....41 W. Main St., Op.

PHILADELPHIA

ALEXANDER, GEO. J.....1629 Spruce St., ALR
ANNON, W. T.....235 S. 15th St., Op.
APPLEMAN, L. F.....308 S. 16th St., Op.
BABBITT, JAMES A.....1912 Spruce St., ALR
BALDWIN, KATE W.....1117 Spruce St., ALR
BALENTINE, P. L.....1524 Chestnut St., Op.
BOEHRINGER, H. W.....1823 Spring Garden St., Op.

BROWN, SAMUEL HORTON.....1901 Mt. Vernon St., Op.
BRUMM, SETH A.....818 Stock Exchange Bldg., ALR
BUTLER, MARGARET F.....1831 Chestnut St., ALR
BUTT, MIRIAM M.....2045 Chestnut St., Op.
CLERF, LOUIS HENRY.....128 S. 10th St., ALR
COATES, GEO. M.....1721 Pine St., ALR
COHEN, HERMAN B.....Spruce and 13th Sts., ALR
COHEN, SAMUEL.....1626 Spruce St., ALR
CREIGHTON, WM. J.....258 S. 18th St., Op.
CROSKEY, J. W.....Aldine Hotel, Op.
DAVIS, J. L.....135 S. 18th St., ALR
DEICHLER, LYN W.....1737 Chestnut St., OALR
DINTENFASS, HENRY.....1305 Spruce St., ALR
EMBERY, FRANK.....4660 Frankford Ave., ALR
ERSNER, M. S.....1915 Spruce St., ALR
FELT, C. L.....Chestnut & 20th Sts., ALR
FOX, L. WEBSTER.....303 S. 17th St., Op.
GLEASON, E. B.....2033 Chestnut St., ALR
GODDARD, H. M.....1531 Spruce St., ALR
GRISCOM, J. MILTON.....2213 Walnut St., Op.
HAWMAN, ERLE G.....4051 N. Broad St., OALR
HEED, C. R.....1205 Spruce St., Op.
HOLLOWAY, THOS. B.....1819 Chestnut St., Op.
HUSIK, D. N.....1610 Spruce St., ALR
JACKSON, CHEVALIER.....128 S. 10th St., L
KAUFMAN, A. S.....1923 Spruce St., ALR
KEELER, J. C.....Medical Arts Bldg., ALR
KRAUSS, FREDERICK.....1703 Chestnut St., OALR
LAESSLE, HENRY A.....The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....Medical Arts Bldg., Op.
LEWIS, FIELDING O.....259 S. 17th St., ALR
LOFTUS, JOHN EDWARD.....605 Medical Arts Bldg., ALR
LUKENS, ROBERT McD.....1308 Hunting Park Ave., ALR
MACKENZIE, ALICE V.....1724 Spruce St., OALR
MACKENZIE, GEORGE W.....1724 Spruce St., ALR
MILLER, E. B.....2008 Walnut St., Op.
MITCHELL, E. K.....704 W. Lehigh Ave., ALR
MOORE, WILLIAM FREDERICK.....255 S. 16th St., ALR
OLSHO, S. L.....235 S. 15th St., OALR
O'REILLY, CHARLES A.....521 Medical Arts Bldg., ALR
PETER, L. C.....N. E. Cor. Chestnut and 20th Sts., Op.
PONTIUS, P. J.....1831 Chestnut St., Op.
POSEY, WM. CAMPBELL.....2049 Chestnut St., Op.
RADCLIFFE, MCCLUNEY.....1906 Chestnut St., Op.
REESE, WARREN S.....230 S. 21st St., Op.
RIDPATH, ROBT. F.....N. E. Cor. 18th and Chestnut St., RL
ROMMEL, JOHN C.....4601 N. Broad St., Op.
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLET, HUNTER W.....230 S. 21st St.,Op.
SCHNEIDEMAN, T. B.....2045 Chestnut St., Op.
SCHWEINITZ, GEO. E. DE.....1705 Walnut St., Op.

SCHWENK, PETER N. K.....1417 N. Broad St., Op.
SENER, WALTER J.....Physicians Bldg., Op.
SHANNON, C. E. G.....135 S. 17th St., Op.
SHEMELEY, WM. G., JR.....1724 Spruce St., OALR
SKILLERN, R. H.....1811 Spruce St.,R
SKILLERN, S. R., JR.....2017 Spruce St., OALR
SMITH, S. McC.....1429 Spruce St., ALR
SPENCER, W. H.....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M.....1130 Spruce St., Op.
STOUT, PHILIP S.....Medical Arts Bldg., ALR
STROUSE, FREDERICK M.....1301 Spruce St., ALR
TUCKER, GABRIEL.....S. W. Cor. 35th and Spruce Sts., B
WAGERS, ARTHUR J.....The Lenox, 13th and Spruce Sts., ALR
WHELAN, G. L.....257 S. 16th St., ALR
WEINSTEIN, M. A.....1309 Spruce St., ALR
WILLIAMS, HORACE J.....5908 Green St., Germantown, ALR
ZENTMAYER, WILLIAM.....1506 Spruce St., Op.

PITTSBURGH

BERNATZ, C. F.....Park Bldg., Op.
BLAIR, W. W.....Diamond Bank Bldg., Op.
CARSON, W. E.....Jenkins Arcade Bldg., Op.
CURRY, GLENDON E.....Westinghouse Bldg.,Op.
DICKINSON, B. M.....5634 Stanton Ave., E. Liberty Sta., ALR
FISCHER, N. A.....623 Union Trust Bldg., ALR
GROSS, JULIUS E.....8030 Jenkins Arcade Bldg., ALR
GUERINOT, A. J.....604 Westinghouse Bldg., OALR
HAGEMAN, JOHN A.....Highland Bldg., ALR
HECKEL, EDWARD B.....Jenkins Arcade Bldg., Op.
KNEEDLER, G. C.....Professional Bldg., ALR
KREBS, ADOLPH.....Jenkins Arcade Bldg., Op.
LINN, J. G.....7026 Jenkins Arcade Bldg., OALR
MCCREADY, J. H.....121 University Pl., ALR
MCKEE, GEO. J.....719 Westinghouse Bldg., ALR
MACLACHLAN, A. A.....May Bldg., ALR
MARKEL, J. CLYDE.....Westinghouse Bldg., Op.
MARSHALL, WATSON.....121 University Pl., ALR
MEANOR, WM. C.....619 Westinghouse Bldg., Op.
MURDOCK, J. F.....Bersemer Bldg., Op.
PATTERSON, ELLEN J.....121 University Pl., ALR
SEEGMAN, SIMON.....Jenkins Arcade, ALR
SHUMAN, G. H.....Park Bldg., Op.
SMITH, STANLEY.....121 University Pl., Op.
STIEREN, E.....626 Union Trust Bldg., Op.
STURM, S. A.....Jenkins Arcade Bldg., Op.
TAYLOR, M. C.....1206 May Bldg., Op.
TODD, GROVER CLEVELAND.....307 Westinghouse Bldg., ALR
TURNER, H. H.....902 Professional Bldg., Op.
VANKIRK, V. E.....626 Union Trust Bldg., Op.
WEIMER, EDGAR S.....332 Lehigh Ave., Op.
WEISSER, E. A.....806 May Bldg., Op.

POTTSVILLE

MOORE, GEO. H.....Schuylkill Bldg., OALR

READING

STOCKLER, J. A.....332 N. 5th St., OALR

RIDGEWAY

MCALLISTER, J. C.....10 S. Mill St., OALR

SCRANTON

CORSER, J. B.....345 Wyoming Ave., Op.
CURTIN, EUGENE A.....703 Connell Bldg., OALR
FREY, CLARENCE L.....529 Vine St., OALR
SULLIVAN, J. J.....228 Scranton Life Bldg., ALR
WILLIAMS, M. M.....148 Adams Ave., Op.

SUNBURY

BECKER, H. M.....49 S. 4th St., OALR

WARREN

BALL, MICHAEL V.....316 Hazel St., OALR

WASHINGTON

MCKENNAN, JAS. W.....514 Washington Trust Bldg., Op.
MCMURRAY, J. B.....Washington Trust Bldg., OALR

WAYNESBURG

STAHLMAN, FRED C.....OALR

WILKES-BARRE

BUCKMAN, E. U.....70 S. Franklin St., OALR
CARR, GEO. W.....54 S. Franklin St., OALR
KISTLER, O. F.....43 N. Franklin St., Op.AR
MCLAUGHLIN, P. A.....71 S. Washington St., OALR
SCHAPPERT, N. L.....57 S. Washington St., OALR
TAYLOR, LEWIS H.....83 S. Franklin St., OALR

YORK

KLINEDINST, JOHN FERD.....220 S. George St., OALR

PHILIPPINE ISLANDS

MANILLA

DEWEY, C. H.....Sternberg General Hospital

RHODE ISLAND

NEWPORT

JACOBY, D. P. A.....106 Touro, Op.AI.

PROVIDENCE

ADAMS, FRANK M.....122 Waterman St., ALR
CAPRON, F. P.....118 Angell St., OALR
HARVEY, N. D.....112 Waterman Ave., OALR
LEECH, J. W.....369 Broad St., OALR
MESSINGER, H. C.....210 Angell St., Op.
MUNCY, WM. M.....162 Angell St., OALR
PORTER, L. B.....199 Thayer St., OALR
SHARP, BENJ. S.....341 Thayer St., OALR

SOUTH CAROLINA

CHARLESTON

KOLLOCK, C. W.....86 Wentworth St., OALR
TOWNSEND, JOHN FERRARS,
85 Wentworth, Cor. St. Phillips St., OALR

GREENVILLE

CARPENTER, E. W.....Professional Bldg., OALR
MAULDIN, L. O.....Chamber of Commerce Bldg., OALR

SOUTH DAKOTA

ABERDEEN

ALWAY, ROBERT D.....423 S. Lincoln St., OALR

HURON

GROSVENOR, L. N.....366 Dakota Ave., OALR

MITCHELL

BOBB, E. V..... OALR

RAPID CITY

WALSH, JOSEPH MARK..... OALR

SIOUX FALLS

GREGG, JOHN B.....231 N. Summit Ave., OALR
KELLER, S. A..... OALR

WATERTOWN

JOHNSON, A. E..... OALR

YANKTON

HOHF, JULIUS.....Clinic Bldg., OALR

TENNESSEE

CHATTANOOGA

HACKNEY, F. J.....630 Volunteer Bldg., OALR
HOGSHEAD, J. M.....602 Georgia Ave., OALR

DYERSBURG

BREWER, JOHN D. ..... OALR

JACKSON

DANCY, ALEXANDER, B. .....First National Bank Bldg., OALR

JOHNSON CITY

JONES, U. G. ..... ALR

KNOXVILLE

KINCAID, J. H. .....421 W. Church Ave., OALR
POTTER, W. W. .....Arnstein Bldg., OALR

MEMPHIS

BLASSINGAME, C. D. .....414 Phys. & Surg. Bldg., ALR
BLUE, J. B. .....Columbian Tower, OALR
ELLETT, E. C. .....Exchange Bldg., Op.
FARRINGTON, P. M. .....Exchange Bldg., ALR
HARRIS, W. ROBIN .....Columbian Tower, ALR
HUGHES, J. A. .....1220 Exchange Bldg., OALR
LEVY, LOUIS. .....1052 Madison Ave., ALR
LEWIS, A. C. .....Exchange Bldg., OALR
MCKINNEY, R. .....1052 Madison Ave , ALR
MINOR, H. F. .....Randolph Bldg., OALR
SELIGSTEIN, MILTON B. .....801 Union Planters Bk. Bldg., OALR
SHEA, J. J. .....1018 Madison Ave., OALR
SIMPSON, W. L. .....Phys. & Surg. Bldg., ALR
STANFORD, J. B. .....1052 Madison Ave., Op.
WATKINS, EDWIN DIAL. .....1412 Exchange Bldg., OALR

NASHVILLE

CAYCE, E. B. .....Medical Arts Bldg., OALR
CULLOM, M. M. .....1119 Bennie-Dillon Bldg., OALR
EZELL, H. C. .....228 Doctors Bldg., OALR
HASTY, F. E. .....420 Lambuth Bldg., OALR
KENNON, W. G. .....Lambuth Bldg., OALR

TEXAS

AUSTIN

BOERNER, MORRIS H. .....Scarborough Bldg., OALR
KEY, SAM N. .....320 Littlefield Bldg., OALR

BROWNWOOD

ANDERSON, W. B. ..... OALR

DALLAS

BETTISON, D. L. .....1102 Medical Arts Bldg., OALR
CARPENTER, E. R. .....708 Medical Arts Bldg., OALR
CARY, E. H. .....4712 Lake Side Drive, OALR
DECHERD, H. B. .....1515 Medical Arts Bldg., OALR
MCLAURIN, J. G. .....1301 Medical Arts Bldg., OALR

McREYNOLDS, JOHN O.....907 Mercantile Bk. Bldg., OALR
NEWTON, F. H.....907 Mercantile Bk. Bldg., OALR
SEAY, D. E.....907 Mercantile Bk. Bldg., OALR
SCHULZE, E. C.....1205 Medical Arts Bldg , OALR
SELLERS, L. M.....Medical Arts Bldg., ALR
STOKES, WM. H.....4105 Live Oak St., Op.
STROUD, EDWARD FRANK.....805 Medical Arts Bldg., ALR

EL PASO

IRVIN, EDGAR H.....321 W. Rio Grande St., OALR
SCHUSTER, FRANK P.....403–1st Natl. Bk. Bldg., OALR
SCHUSTER, S. A.....1st Natl. Bank Bldg., ALR

FORT WORTH

HOWARD, EDWARD L.....First Natl. Bank Bldg., OALR
SCHENCK, C. P.....604 Ft. Worth Natl. Bank Bldg., OALR

HOUSTON

DAILY, LOUIS.....1117 Med. Arts Bldg., OALR
DAILY, R. K.....1117 Med. Arts Bldg., OALR
FOSTER, J. H.....1306 Walker Ave., ALR
GOAR, EVERETT LOGAN.....1300 Walker Ave., Op.
HADEN, HENRY C.....Niels Esperon Bldg., Op.
ISRAEL, NORMA E.....2001 Niels Esperon Bldg., OALR
ISRAEL, SIDNEY.....2001 Niels Esperon Bldg., ALR
RALSTON, WALLACE.....1300 Walker Ave., Op.

LONGVIEW

HURST, V. REEVES..... OALR

SAN ANTONIO

ADAMS, ELDRIDGE S.....Moore Bldg., ALR
ATKINSON, D. T.....827 Med. Arts Bldg., OALR
BURLESON, J. H.....Eye, Ear, Nose & Throat Hospital, OALR
GWINN, G. E.....704 Central Trust Bldg., OALR
PARRISH, MAJ. ROBT. E., M.C., U. S. A...125 Herbert Ave., ALR

TEMPLE

McREYNOLDS, G. S.....State Bank Bldg., OALR
WOODSON, J. M.....Cor. Ave. G and Fifth, OALR

TEXARKANA

FULLER, T. E.....Texarkana Natl. Bank Bldg., OALR
MANN, R. H. T.....State Natl. Bank Bldg., OALR

TYLER

VAUGHN, E. H..... OALR

WACO

AYNESWORTH, H. T.....1307 Amicable Bldg., OALR

UTAH

OGDEN

HETZEL, C. C. ..... Eccles Bldg., OALR

PROVO

MERRILL, H. G. ..... 33 East 2nd St., OALR

SALT LAKE CITY

DONOHER, WM. D. ..... Boston Bldg., OALR
HAMPTON, ROBT. H. ..... 804 Boston Bldg., ALR
LE COMPTE, E. D. ..... 627 Boston Bldg., OALR
NEHER, EDWIN MANSON ..... Boston Bldg., Op.
STAUFFER, FRED ..... 133 1st Ave., OALR

VERMONT

BURLINGTON

ARNOLD, FRANCIS J. ..... 182 Pearl St., OALR
BROWN, EARLE TOWLE ..... 30 Church St., OALR
PHELPS, H. S. ..... 322 S. Prospect St., ALR

RUTLAND

MARSHALL, GEORGE GUERIN ..... Gryphon Bldg., OALR

VIRGINIA

RICHMOND

GATEWOOD, E. T. ..... Professional Bldg., ALR
HILL, EMORY ..... Professional Bldg., Op.
HUGHES, T. E. ..... 1000 W. Grace St., ALR
MILLER, CLIFTON M. ..... Stuart Circle Hospital, OALR

ROANOKE

GARRETT, J. R. ..... Mt. Trust Bldg., OALR
GILL, E. G. ..... McBain Bldg., ALR
HAMLIN, F. EUGENE ..... 218 Shenandoah Life Bldg., ALR
YOUNG, CHARLES A. ..... Gill. Mem. E. E. and Throat Hospital, Op.

WINCHESTER

MCGUIRE, H. H. ..... 105 N. Braddock St., OALR

WASHINGTON

ABERDEEN

MAXEY, E. E. ..... 609 Becker Bldg., OALR

BELLINGHAM

VAN KIRK, FRANK J. ..... Bellingham Natl. Bank Bldg., OALR

SEATTLE

ADAMS, FRED'K.....1114 Boylston Ave., OALR
CHASE, E. FRANK.....1008 Cobb Bldg., OALR
DOWLING, J. T.....Virginia Mason Hospital Bldg., OALR
HOFFMAN, W. F.....817 Summit, OALR
SEELYE, W. K.....742 Stimson Bldg., OALR
STILLSON, H.....Seabord Bldg., OALR
WURDEMANN, H. V.....Cobb Bldg., Op.

SPOKANE

ROTT, O. M.....Paulsen Bldg., OALR
VEASEY, C. A.....Paulsen Bldg., OALR
VEASEY, C. A., JR.....404 Paulsen Bldg.

TACOMA

CAMERON, WM. G.....1404 Puget Sd. Bk. Bldg., OALR
DAVIES, J. S.....704 St. Helen's Ave., OALR
HOWE, A. W.....1616 Washington Bldg., OALR
WHEELER, ERNEST C.....Rust Bldg., OALR

WEST VIRGINIA

CLARKSBURG

LOUCHERY, D. C.....212 W. Main St., OALR

FAIRMONT

JOHNSON, H. R.....512 Walnut Ave., OALR

HUNTINGTON

MOORE, T. W.....1050 Fifth Ave., OALR

MORGANTOWN

WYLIE, C. B.....293 High St., OALR

WHEELING

FAWCETT, I.....75 12th St., OALR
JONES, EDMUND LLOYD.....Wheeling Clinic, OALR
TOMASSENE, R. A.....Wheeling Bk. & Trust Bldg., OALR

WISCONSIN

APPLETON

BROOKS, E. H.....108 W. College Ave., OALR
RECTOR, A. E.....112 W. College Ave., OALR

EAU CLAIRE

PAYNE, A. L.....Opera House Bldg., OALR

FOND DU LAC

FOLSOM, W. H.....395 E. Division St., OALR

GREEN BAY

NADEAU, E. G. ..... Bellin-Buchanan Bldg., OALR

JANESVILLE

PEMBER, AUBREY H. ..... 500 W. Milwaukee St., OALR

KENOSHA

ASHLEY, T. W. ..... 5711 Eighth Ave., OALR

LA CROSSE

BRADFIELD, J. A. L. ..... 1515 Main St., OALR
DOUGLAS, FREDERICK ALEXANDER. ..... 303 State Bank Bldg., OALR
SMITH, D. S. ..... State Bank Bldg., OALR

MADISON

BARLOW, ROY A. ..... ALR
DAVIS, F. A. ..... 122 W. Washington Ave., OALR

MANITOWOC

ANDREWS, M. P. ..... OALR

MARINETTE

MAY, J. V. ..... 1701 Main St., OALR

MARSHFIELD

COPPS, LYMAN ALDEN ..... OALR

MILWAUKEE

BEEBE, C. S. ..... 509 Wells Bldg., Op.
BLACK, N. M. ..... 1240 Wells Bldg., Op.
CHAPMAN, V. A. ..... Colby-Abbott Bldg., OALR
GORDON, J. S. ..... 141 Wisconsin St., OALR
GROVE, W. E. ..... 120 E. Wisconsin Ave., OALR
HEEB, HARRY J. ..... 204 Grand Ave., OALR
HIGGINS, S. G. ..... Wells Bldg., OALR
HITZ, H. B. ..... Goldsmith Bldg., ALR
MULSOW, J. E. ..... 230 Wisconsin Ave., OALR
RICE, R. H. ..... 331 Grove St., OALR
SEAMAN, GILBERT E. ..... 407 Wells Bldg., OALR

RACINE

HOLMES, BENJAMIN H. ..... 435 Baker Blk., OALR

SUPERIOR

HOVDE, A. G. ..... Board of Trade Bldg., OALR
MYERS, ELMER A. ..... 407 Board of Trade Bldg., OALR
SHASTID, T. H. ..... 10th Ave. Med. Bldg., Op.
SMITH, R. C. ..... Board of Trade Bldg., OALR

WAUSAU

SCHLEGEL, HERMAN T. ..... 501 Third St., OALR
WILLARD, L. M. ..... 520 Third St., OALR

WYOMING

CHEYENNE

BECK, FREDERICK L.....408 Hynds Bldg., OALR
STRADER, GEO. L.....408 Hynds Bldg., OALR

AFRICA

NATAL

DURBAN

JOHNSON, LINDSAY.....Britannia Bldg.

CANADA

ALBERTA

EDMONTON

BROWN, W. HAROLD.....Bank of Montreal Bldg., OALR

BRITISH COLUMBIA

VANCOUVER

BOUCHER, R. B.....Birks Bldg., OALR

VICTORIA

KEYS, MORTON J.....307 Jones Block, OALR

MANITOBA

BRANDON

MCDIARMID, H. O.....35 Clement Blk., OALR

NEW BRUNSWICK

ST. JOHN

CHIPMAN, L. D. V.....42 Coburg St., OALR

NOVA SCOTIA

HALIFAX

CUNNINGHAM, ALLAN R.....260 Barrington St., OALR

ONTARIO

HAMILTON

ARRELL, W.....317 Main St., E., OALR
MACFARLANE, P. B.....152 James St., S., OALR

OTTAWA

FRASER, W. G.....52 Nepean St., OALR

PETERBOROUGH

BUCHANAN, NORMAN D.....479 Water St., OALR

TORONTO

DAVIES, T. A.....578 Sherborne St., OALR
GOLDSMITH, P. G.....84 Carlton St., ALR

HILL, C. E.....160 Bloor St., W., Op.
MCCULLOCH, R. J.....102 College St., OALR
MCFARLANE, MURRAY.....190 Bloor St., E., OALR
MCKELVEY, A. D.....160 Bloor St., W., ALR
PENTECOST, R. S.....184 Bloor St., OALR
RISDON, E. F.....30 Avenue Rd., ALR
WRIGHT, WALTER WALKER.....143 College St., Op.

QUEBEC

MONTREAL

BALLON, D. H.....107 Crescent St., ALR
BIRKETT, H. S.....1190 Mountain St., ALR
BOUSQUET, J. P. E.....26 Sherbrooke St., W., OALR
LASALLE, ALBERT.....3439 St. Denis St., OALR
LUNDON, A. E.....119 Crescent St., ALR
MCKEE, S. HANFORD.....158 Crescent St., Op.
MACMILLAN, J. A.....1429 Stanley St., Op.
RAMSEY, G. S.....374 Mountain St.
ROGERS, J. T.....758 Sherbrooke St., W., ALR
ROSENBAUM, J.....206 Bishop St., Op.
WHITE, E. H.....756 Sherbrooke St., W., ALR

CZECHO-SLOVAKIA

PRAGUE

ELSCHNIG, ANTON.....Ferdinandstrasse, 10

ENGLAND

LONDON

DUNDAS-GRANT, SIR JAMES.....148 Harley St., W. 1.
ELLIOT, LT.-COL. R. H., 54 Welbeck St., Cavendish Sq., W.
GRAVES, BASIL.....35 Weymouth St., W.
KIRKPATRICK, LT.-COL. HENRY..... 54 Welbeck St., W. 1
LAWFORD, J. B.....27 Weymouth St., Portland Pl.
LISTER, SIR WILLIAM T.....24 Devonshire Pl., W. 1

HOLLAND

LEIDEN

VAN DER HOEVE, PROF. J.....Rijnsburgerweg 6A

SCOTLAND

GLASGOW

GRAY, ALBERT A.....

SWITZERLAND

ZURICH

NAGER, PROF. F.....Freiestrasse 20

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